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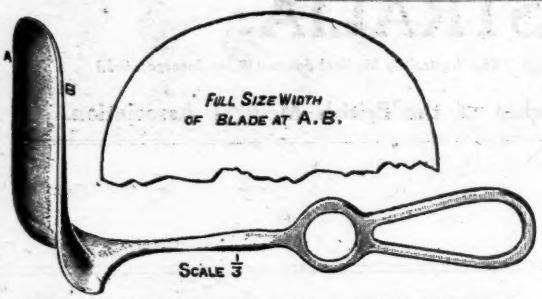


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# THE MEDICAL JOURNAL OF AUSTRALIA.

VOL. II.—4TH YEAR. SYDNEY: SATURDAY, AUGUST 25, 1917. No. 8.

## THE BROKEN HILL EPIDEMIC.[1]

By G. H. Burnell, M.B., B.S. (Adel.),
*House Surgeon, Broken Hill Hospital.*

As a junior member of the profession, it is with some diffidence that I bring forward my account of the recent epidemic at Broken Hill. The ætiology of the disease is very obscure, and my reason for bringing to your notice an unfinished investigation is that the case mortality of the disease is very high (80%), and that I am hoping that a full discussion will bring to light some valuable suggestions as to further methods of research, and turn the attention of abler men than myself to this problem.

There have been 15 cases of this disease, spread over the period from February 28, 1917, to April 9, 1917. Twelve patients have died. There have also been several deaths after a short illness of a day or two in some of the outlying townships, and from the meagre information available it appears probable that they also were affected by this disease. However, I propose to confine myself for the present to the cases we have seen.

### Ætiology.

In age the patients range from 5 years to 61 years. All were males. There has not been even a suspicious case in a female.

Six of the ten men have been miners, but this in Broken Hill is not remarkable, as the greater part of the male adult population follow that occupation.

The meat, milk and water supply have been carefully investigated by the City Sanitary Inspectress, who can find no common source of infection, nor do the patients appear to come from specially insanitary surroundings.

The cases have been scattered fairly evenly throughout the city, and, as has been mentioned above, similar cases have probably occurred in the country surrounding Broken Hill.

The only evidence bearing on possibility of infection from contacts is the fact that a man, J.T.H., met the patient Ch. in Menindie on March 20, 1917, and looked after him for the night. The next day he (J.T.H.) went to a station some 80 miles away, where he worked as a cook. He lived in a hut with a man, Me. Me. was taken ill on April 6, and died in the hospital on April 8.

Even if this is more than a coincidence, we do not know whether infection was by contact or by food contamination, as the suspected person is a cook.[2]

### Symptoms.

The patient has a headache for a day or two before seeking treatment. This is not confined to any particular part of the head, is not very severe and does not prevent sleep.

There is a feeling of great weakness, and some patients complain of loss of power in the limbs, but there is never any actual paralysis.

Vomiting has been present in five cases out of fifteen. It has no relation to meals, and is not very severe or persistent. There is no epistaxis.

After one or two days the patient passes into a condition of semi-unconsciousness, in which his mental condition is very unusual and striking. He lies with both eyes open, apparently in very little pain, and may appear to take an intelligent interest in what is passing around him. On request he will promptly put out his tongue and perform simple movements, but he does not appear to be able to grasp longer or complicated sentences. He does not speak at all, and there is very little restlessness. A very marked feature of this mental condition is a peculiar resistiveness, by which I mean that the patient gently, but firmly, resists any manipulations one may attempt to make with his limbs. He does not struggle, but merely stiffens his whole body.

The temperature ranges from 38.3° C. to 41.7° C. (101° to 107° F.) in our cases, but usually runs about 39.5° to 40° C. (103° or 104° F.), with irregular remissions of a degree or two.

The pulse varies in rate from 70 to 120. It is full and strong, and remains so even till death. It has been noted in case after case that the pulse remains good, even when the patient is obviously moribund.

The tongue is coated all over with a thick brown fur.

The respiration rate is at first not increased.

The bowels are constipated, enemata or aperients being usually necessary.

The pupils are equal, and the reaction to light constantly present.

Head retraction and Kernig's sign are sometimes present temporarily. I emphasize the word temporarily, for a striking feature of the disease is the transient nature of all the symptoms. In the morning one may find the patient with marked head retraction, opisthotonos, rigid limbs, or perhaps general clonic convulsions, with Kernig's sign well marked, and absent knee-jerks, while an hour later he may be lying quietly in bed with none of these symptoms.

As a rule the reflexes, both superficial and deep, are present and not exaggerated.

There is no splenic enlargement, no *tâche cérébrale* and no rash on the body.

The urine does not usually contain any albumin (three cases out of fifteen).

There is said to be a distinctive odour about these cases, but having very little sense of smell, I am unable to corroborate that.

This is the usual condition of a patient on the day of his admission.

[1] Read at a Meeting of the South Australian Branch of the British Medical Association on April 26, 1917.

[2] Since writing the above it has been ascertained that J.T.H. also was in the company of another patient, J.H., on March 19, 1917. J.H. was taken ill on April 5, 1917, and died on April 11.

The next day the mental condition has changed, and the patient lies in a state of profound unconsciousness, passing his stools and urine under him. As before, the head retraction and clonic convulsions may come and go. Some of the patients have had a *risus sardonicus*, but this too is only transient.

A very common symptom at this stage is difficulty in swallowing, and we have had to resort to the œsophageal tube for feeding purposes.

Cyanosis and respiratory embarrassment now appear, and increase steadily until death. The first sign is the increase in depth of the respiratory movements. The respiration assumes a Cheyne-Stokes' rhythm, and is very laboured. Later on, and for the last 24 hours before death, it increases in frequency, ranging usually from 30 to 50. In one case the patient frothed at the mouth very freely for 36 hours before death.

The death is a typical respiratory death, there being no spasm of the respiratory muscles, but, I take it, a paralysis or exhaustion of the respiratory centre. There is a tendency for the temperature to rise just before death.

It will be interesting to refer to the notes of a case treated by a nurse at White Cliffs, there being no medical man within reach. Their interest lies in the fact that the nurse at this time had neither seen nor heard of our cases.

**Symptoms of Patient When First Seen by Nurse.**

Visited patient about 7.20 on the evening of April 7. Temperature 103.4°, pulse 104; face flushed, tongue furred; patient seemed delirious, spoke rationally at intervals, complained of pains in loins. Was delirious and got out of bed during the night. I visited patient between nine and ten o'clock on Sunday morning, asked him how he was; he said "Better"; looked very ill; tongue had band of fur on either side, black line down centre; lips dry and cracked. Breathing rapid. I did not see the patient in early stage, but was told he vomited and had pains in neck, limbs and complained for several days of headache. Patient kept about until Saturday morning. He was delirious all day Sunday and Sunday night; had to be kept in bed by being held. He moaned loudly all night; only slept for short intervals; breathing easy when dozing, rapid when awake. Kept throwing his legs about. Left arm stiff from elbow; eyes glazed in early part of illness, later inflamed; clear watery discharge from them. Bowels open with aperient. Skin on forehead and round neck acted slightly during the night. Temperature ranged from 103.4° to 104°; pulse 104 to 112; respiration 36. On Monday morning patient showed pneumonic symptoms.* Skin acted very freely. Patient appeared to be in great pain, closed mouth firmly and knitted his brows. He did not speak from about 6 p.m. on Sunday morning. He died at five minutes to three on Monday afternoon. The temperature reached 106° just before death.

The duration of the disease in fatal cases is from three to five days from the first onset of headache. The non-fatal cases have not reached the stage of respiratory embarrassment, and in them the marked feature is the profound mental change. In two of them, even now, after some weeks of apyrexia, delusions are still present. The patient will talk quite rationally for some minutes, and then say something quite irrational.

Since the above was written, one patient (J.E.) has recovered mentally, but only after eight weeks, and the other (C.) is still in a state of sub-acute mania, but is, I think, gradually improving mentally.

* Probably the cyanosis.

**Morbid Anatomy.**

Post-mortem examinations have been carried out in five cases, but in only three have we been able to examine the brain. In two cases the meninges were normal. In the third slight injection of the pia mater was noted. There was no pus in the pia-arachnoid in any case. Marked œdema of the brain was present in one case, the fluid continuing to drip from the organ for some time after it had been removed. No microscopical sections of the brain or cord were made.

No post-mortem clot was present in the heart, the blood remaining very fluid, and in one case the heart completely emptied itself through the cut cerebral vessels.

The stomach and liver were normal. There was no enlargement of the spleen.

The kidneys were normal in three cases, in which there had been no albuminuria. There was a definite chronic interstitial nephritis in one case with albuminuria, while in the other, except for some congestion, the kidneys were macroscopically normal.

The suprarenals and pancreas were normal in the four cases examined.

In four of the five cases there was definite injection of the mucosa of the jejunum and ileum, the amount varying in the different cases, but being always more marked in the jejunum than the ileum. There was no enlargement of Peyer's patches, and no ulceration. The large bowel was normal in each case.

**Differential Diagnosis.**

The cases have been variously diagnosed as:—

(1) Tubercular meningitis,
(2) Cerebro-spinal meningitis,
(3) Uræmia, and
(4) Tetanus.

At times these cases have features in common with each one of the conditions mentioned, but here again I would emphasize the evanescensce of each symptom.

Other conditions which would need to be considered are:—

(5) Strychnine poisoning, and
(6) Severe influenza with meningeal involvement.

The rapid course of the disease, together with the absence of small lymphocytes in the spinal fluid, and also the absence of post-mortem findings serve to exclude tubercular meningitis. As regards cerebro-spinal meningitis, the differential diagnosis is of some considerable importance, both from the point of view of prognosis and from that of treatment. This is especially the case at present, when the meningitis "season" is re-approaching.

I may say here that in most of the typical cases of cerebro-spinal fever we have been able to isolate the meningococcus from the spinal fluid.

The best means of diagnosis is by lumbar puncture. There is invariably a complete absence of any polymorpho-nuclear leucocytes from the spinal fluid.

Uræmia is excluded by the fact that in the great majority of cases the urine has been quite free of albumin.

Similarly, both tetanus and strychnine poisoning are excluded, as, although some patients have had some twitchings, there have been others in whom no sign of twitching was present throughout the disease, and, again, even in those in whom the twitchings were present, they disappeared usually for 24 hours before death.

The affection was unlike influenza in that there was evidence of affection of the air passages in only one case out of fifteen. In addition, the usual cardiac and intestinal symptoms of influenza were lacking. There was not the evidence of infection from patient to patient, as is usual in influenza; there were only two patients from one house. Lastly, the case mortality is too high, and there have not been any mild cases of influenza in the district, as one would have expected.

One patient (F.N.) was unlike the others. From the very onset his condition resembled that of enteric fever, with rose spots and a palpable spleen. Later on his blood serum gave a positive Widal reaction. I can only suggest that in his case there was a double infection.

Lumbar puncture has been carried out in nearly every case. The fluid obtained was quite clear and under slight pressure, but its withdrawal did not in any case apparently have any favourable effect, and in the later cases of the series it was given up.

The spinal fluid was examined microscopically in nine cases (after incubating it for 24 hours at 37° C.), and in only one case (G.T.) was there any appreciable number of cells, and they were small lymphocytes. There has been no case with an excess of polymorpho-nuclear leucocytes.

Leucocyte blood counts were made in five cases—two fatal and three non-fatal. The two former showed a leucocytosis of 19,000 and 20,000. In both cases a differential count showed that the various kinds of leucocytes retained their normal proportion to each other. In the three non-fatal cases the white count was between 7,000 and 8,000 per cubic millimetre. In these three Widal tests were carried out, as well as in control cases of suspected enteric fever. One of the three (F.N.) gave a positive Widal reaction, but, as mentioned above, he was suffering from a clinically typical enteric fever.

The bacteriological findings in the series of cases, although not definite, are nevertheless very interesting. The methods adopted were: First, the spinal fluid was incubated for 24 hours at 37° C., and the pellicle which forms in it examined, after straining by Ziehl-Neelsen and Gram's methods. Second, cultures from this pellicle were made on agar tubes.

In the earlier cases these were the sole means of examination, but I thought that the chances of accidental infection, while waiting for the fluid to drip into a test tube, were too great, so I then tried culturing blood on agar slants and in peptone broth. About 1½ c.cm. of blood were taken from a vein with a hypodermic needle (after careful cleansing of the skin) and ½ c.cm. squirted over the agar slant, the remainder being squirted into tubes or bottles containing 10 to 15 c.cm. of broth.

Out of ten cases so treated, in seven bacillus No. 1 was obtained on the agar slant, no growth in two cases, and in one case bacillus No. 2. (See Chart I.)

The broth culture was only tried in five cases, and in these bacillus No. 1 was obtained in three, bacillus No. 2 in one case, and in one case no growth.

### Bacteriology.

Bacillus 1 developes on the agar slant in numerous small, translucent colonies. In 36 hours from sowing they are about half the size of a pin's head, and in the course of the next 24 hours they grow to about twice the size of a pin's head.

On agar the subcultures grow readily, giving a colony rather like a typhoid bacillus, except that the edges are more inclined to be "feathery." The surfaces of young subcultures are smooth, but in older ones are distinctly granular.

It is a short bacillus, and assumes several varied forms, being at one time coccal, at others coccobacillary or pure bacillus. It often occurs as a diplobacillus. It is non-motile, and does not stain by Gram's method. It does not form spores. In broth it causes first a uniform turbidity, but after several days a fairly thick membrane developes on the surface, this membrane having a distinct honey-combed appearance.

It grows fairly well on untreated potato, the growth having well-defined edges and being slightly darker than the potato.

It does not liquefy gelatine.

*Fermentation Tests.*—For the first three days it produces no change in either glucose, lactose, saccharose, dulcite, mannite or litmus milk. After that time the findings are not quite constant, as in two cases it produced slight acid in glucose, but never any gas.

Bacillus No. 2 is a large Gram-positive organism. It has square ends and occurs in short chains. It grows readily on any media. In broth it causes a general turbidity, and forms spores freely, the spores being central in position and not causing any bulging of the bacillus.

It is probably the *bacillus subtilis*, and is probably an accidental infection. It is freely present in the Broken Hill water supply.

It will be noticed that in ten out of the thirteen cases on which bacteriological work was done bacillus No. 1 was isolated either from blood or cerebro-spinal fluid.*

It was also isolated in almost pure culture from the lung of one patient (A.A.) removed at autopsy.

Experimental inoculations were tried, but without success.

(1) On March 23 two guinea-pigs were inoculated subcutaneously, one with bacillus No. 1 and the other with bacillus No. 2. They are both well.

(2) On March 28 a rabbit was inoculated intra peritoneally with bacillus No. 1. It was stolen the night after inoculation.

* It is interesting to note that typhoid bacilli have been found in the cerebro-spinal fluid of cases showing meningeal symptoms.—Stitt.

(3) On April 14 rabbit inoculated intravenously with bacillus No. 1. It is still alive.

(4) On April 23 guinea-pigs fed on food containing bacillus No. 1 are still alive.

*Agglutination Tests.*—I also endeavoured to ascertain whether the serum of the three recovering patients would agglutinate bacillus No. 1. Dilutions of their serum, together with that of two control enteric fever patients, were made, a strength of 1 in 40 being used. This proved negative in all five cases. I therefore tried dilutions of 1 in 10, but again without success.

These bacteriological findings are far from being conclusive, but I consider that they are of sufficient importance to warrant further investigation, and it is with that end in view that I place them on record.

It has been suggested that the bacillus No. 1 is merely a secondary infection superimposed upon some other unrecognized condition, and thus passing a more or less saprophytic existence, and in view of the negative nature of the inoculation experiments, this seems quite probable. I have endeavoured to eliminate all sources of error, and the personal factor would appear to be excluded by the fact that this same bacillus was obtained by Dr. J. B. Cleland from one of my cases, and was also obtained by Dr. Birks from another patient, who came into hospital and died during my temporary absence.

This organism, judged from both its morphology and fermentative characteristics, apparently belongs to the group of hæmorrhagic septicæmias or pasteurelloses.

"The chief member of this group, and the only one from which man has been known to suffer, is the *bacillus pestis* of plague. Other pasteurelloses are those of chicken cholera, swine plague, mouse septicæmia, rabbit septicæmia and the septicæmia of cattle. It is a widely distributed group, and may include saprophytic organisms as well as those noted for their virulence."—Stitt.

All observers are agreed as to the great tendency for members of this group to undergo involution changes, indeed, their polymorphism is more marked than is the case with any other group of organisms.

In this connexion it is interesting to note the work which has been done on this group at the Lister Institute (*Journal Pathology and Bacteriology*, 1915-1916), and at the Royal College of Physicians, Edinburgh. From the latter place J. P. McGowan writes: "We have observed variations of biological activity among different strains of the same organism, derived from different sources, whether it be that of chicken cholera, swine plague, or sheep septicæmia. These variations (speaking more especially with regard to sugar reactions) may consist in a lack of power in some strains to ferment any of the sugars; on the other hand, they may act upon them in many different combinations, changing some and leaving other unchanged. Such inconstancy of action would appear to account in some measure for the hazy and altogether indefinite description of the reactions of such organisms on the carbohydrates as is usually given in text-books. Not only does this group . . . . vary greatly with regard to its biological activity, but it also varies greatly in its power of altering its shape."

I have dwelt at some length on this change in biological activity as an attempt to explain the presence of the bacilli in the blood of the patient F.N., who showed none of the clinical signs of the disease, and who also undoubtedly had enteric fever. Possibly there was some antagonism between the two infections.

### Treatment.

With an 80% case mortality it is evident that our treatment left much to be desired.

The following measures were adopted:—

(1) Isolation of the patients.

(2) Venesection, withdrawing 570 to 850 c.cm. (20 to 30 ounces) of blood.

(3) Lumbar puncture and injections of anti-meningococcal serum into the spinal theca. In the later cases this was discontinued, as it was of no benefit.

(4) Intravenous injection of saline solution (also subcutaneous).

(5) Intravenous injection of eusol in 100 c.cm. doses.

(6) Salol in 0.6 gm. (10 grains) doses, dissolved in paroleine, was given by mouth three times a day. This was suggested by finding post-mortem the injection of the upper small bowel.

(7) Salicylate of soda in 0.9 gm. (15 grains) doses three times a day.

(8) Strychnine hypodermically.

As death appeared to be due to respiratory failure, strychnine would appear to be indicated, but in view of the irritative condition of the patient's nervous system, one hesitated to use it. However, I tried it in one case, but without any appreciable benefit.

In conclusion, I would point out that whether the bacteriology be accepted or not, we have a symptom-complex which does not conform to any of the ordinary diseases, and I am hoping that other cases may be recorded which will lead to an advance in the study of its ætiology and treatment.

I desire to record my heartiest thanks to Dr. Melville Birks, the Superintendent of the Broken Hill Hospital, for valuable suggestions and encouragement in the work, and also to Dr. J. B. Cleland, of Sydney, who did the more technical part of the bacteriological investigation and finally succeeded in "placing" the organism.

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Since writing the above a further case of the disease has occurred, having rather interesting features, in that a new line of treatment was tried, *viz.*, injection of the serum of a patient who had recovered from the disease (C. of my series, the patient who was maniacal for a period of some months, but who is now (June 4, 1917) quite rational). Treatment was begun at a stage when the patient was apparently going rapidly downhill, just as nearly all the other had done; but from the time of the first in-

jection she rapidly improved, and is now ready for discharge.

Whether this is a case of "*post hoc*" or "*propter hoc*" I am unable to say, but it suggests interesting possibilities as to the serum treatment of this new disease. The other interesting thing is that this patient was a female, the first of our series.

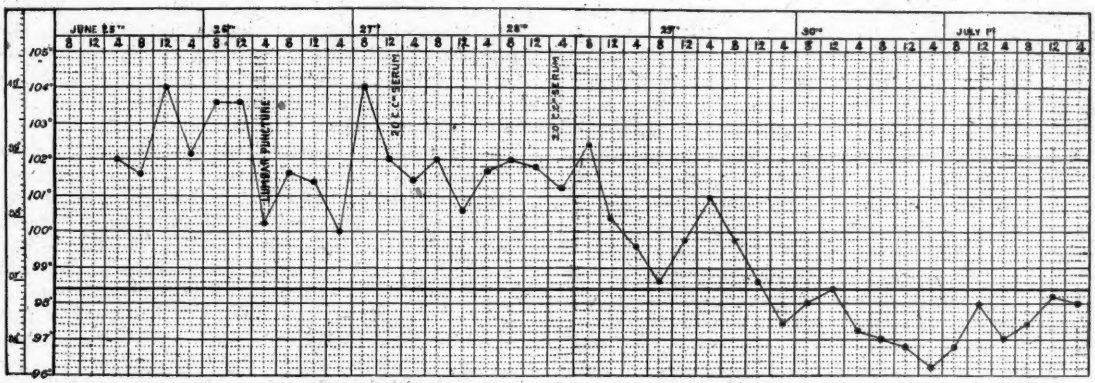
V.E.R., æt. 12 years, a female, was admitted on June 25, 1917. The child had been ill for two days. The illness began with headache, followed quickly by pains in the back and vomiting. Since shortly after the onset the child had not spoken, and would not reply to questions, although she was apparently quite rational. The bowels have been constipated, but acted just before admission. The previous history was good.

The blood count gave the following result:—

| | |
|---|---|
| Red cells | 4,200,000 |
| White cells | 25,000 |
| Differential count:— | |
| Polymorpho-nuclear leucocytes | 83% |
| Large lymphocytes | 9% |
| Small lymphocytes | 6% |
| Transitional cells | 1% |
| Eosinophile cells | 1% |

The polymorphs all showed a circular arrangement of their nuclei. The significance of this I do not know.

Cultures of the blood and spinal fluid were taken. In addition, 1.5 c.cm. of fresh blood were injected into the peritoneal cavity of a guinea-pig; but all these measures gave negative results.




On admission, the temperature was 38.9° C. (102° F.), the pulse 132, regular, of good volume and tension, and the respirations 34. The child was semi-rational; she would not answer questions, but put out her tongue when requested. She was very restless, and unless closely watched got out of bed and walked about. There was no head retraction.

The pupils were equal, and reacted to light and accommodation. There were no abnormal signs in the heart and lungs. There was no retraction in the abdomen, and no rash. The knee-jerks were absent. Kernig's sign was absent. Urine, specific gravity 1,030, a trace of albumin.

Under chloroform lumbar puncture was performed, and 30 c.cm. of perfectly clear fluid were removed. Examined under the microscope this showed only a few lymphocytes.

June 27, 1917.—The condition was the same, except for a slight increase of drowsiness; 20 c.cm. of "C.'s" serum were given subcutaneously.

June 28, 1917.—The mental condition was better. The temperature was still elevated; 20 c.cm. of "C.'s" serum were injected subcutaneously.

June 29, 1917.—The patient was distinctly better, and the temperature falling.

July 1, 1917.—She was doing well.

July 5, 1917.—Discharged.

I had intended giving the serum intravenously and intrathecally as well, but the case was urgent, and it took several days to obtain the serum sufficiently clear for intravenous injection. I have now obtained it clear, and will use it intravenously should another case occur here.

**Table Showing Methods Adopted in Isolating the Organism.**

| | Patient's Initials | | | | | | | | | | | | | Controls. | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | K.T. | G.T. | G.B. | R.B. | C. | A.A. | J.B. | A.M. | F.W. | F.N. | B.M. | J.H. | J.McG. | a. | b. | c. G.H.B. | d. Sister | e. H. | f. Tm. |
| C'bro-Spin'l Fluid | I. II. | I. II. | II. | II. | — | I. | * | * | I. | — | — | — | * | — | — | — | — | — | — |
| Blood on Agar | — | — | — | II. | I. | * | I. | I. | I. | I. | I. | * | I. | * | * | * | * | * | * |
| Blood on Broth | — | — | — | — | I. | * | I. | II. | — | — | — | — | I. | — | — | — | — | — | — |
| C.S.F. on Agar | I, II. | I. II. | II. | II. | — | I. II. | — | — | — | — | — | — | — | — | — | — | — | — | — |
| Leucocytes | — | — | — | — | 8,000 | — | 7,800 | 20,000 | 19,000 | 7,000 | — | — | — | — | — | 10,000 | — | — | — |
| Widal | — | — | — | — | —ve | — | —ve | — | — | +ve | — | — | — | +ve | —ve | —ve | — | +ve | +ve |
| Agglutination Against Bacillus I.— | | | | | | | | | | | | | | | | | | | |
| Dilution 1 in 40 | — | — | — | — | —ve | — | —ve | — | — | —ve | — | — | — | —ve | —ve | — | — | — | — |
| Dilution 1 in 10 | — | — | — | — | —ve | — | —ve | — | — | —ve | — | — | — | —ve | —ve | — | — | — | — |

The control cases were four suspected enterics in their first week, together with the sister in charge and myself.

I. is the small Gram-negative non-motile bacillus. II. is the large Gram-positive motile bacillus (*subtilis*).

* No growth.

## SOME NOTES ON CASES OF PERFORATED GASTRIC AND DUODENAL ULCERS.[1]

By D. Kelly, M.B., B.S.,
*Senior Honorary Surgeon, Mater Misericordiae Hospital, North Sydney.*

The statement made by Collinson (*Journal of American Medical Association*, October 3, 1914) that perforation of a gastric or duodenal ulcer is, with the exception of appendicitis, the most frequent acute abdominal lesion, does not fit in with my experience of acute abdominal conditions, nor do the records of the Sydney hospitals agree with it.

In the last five years I have only seen ten cases of perforation of the stomach or duodenum, eight of which were under my own care. All of these were in males, the youngest of whom was 24 years and the oldest 49 years. The two youngest were thin subjects, the others were in good condition, though none were fat.

There was a definite history of chronic indigestion in every patient. The symptoms, varying from fullness and discomfort to quite severe pain, occurred in some a quarter of an hour, and in others up to two or three hours, after the ingestion of food. Belching of wind was a constant symptom. Six of them noted that there were times when their indigestion was much better. One said there had been no relief in two years, and one, the youngest, stated that for two months before perforation occurred, he had been free from discomfort. All had tried some means of relieving their symptoms, and found that drinking hot water and taking bicarbonate of soda gave the most satisfaction.

The induction of vomiting in three of the cases gave relief, but in none was vomiting a symptom, nor did any ever vomit blood or ever notice tarry motions.

The causes determining the perforation varied. In three perforation occurred in the early morning hours, after light meals the night before. One was on a diet of Benger's food and milk at the time of perforation, which occurred whilst lifting a weight two hours after taking a meal. One had a light meal at mid-day, and perforation occurred four hours later. The oldest patient of the series, annoyed at always having to study his diet and even then suffering, had a big meal one night of brawn, onions, bread and butter, and two cups of tea. He then went to sleep, and was awakened three hours later with intense pain in the pit of his stomach.

The youngest, who had been free from symptoms for two months, stated that he had had three or four glasses of beer, and was helping a friend home when he was suddenly seized with violent pains in the belly.

Only one vomited after perforation had taken place; thus nature attempted to protect the injured organ by practically paralysing it. Sudden, acute abdominal pain was the first symptom in all cases. In seven the acute pain was first felt in the pit of the stomach, and one stated that the pain was first felt in the lower right quadrant.

An absolute board-like hardness characterized the abdominal wall, and the facies, although giving the expression of severe pain, never presented the pinched and anxious look of acute peritonitis.

One patient was seen by his doctor at 6 p.m., and was given a hypodermic injection of 0.0125 gm. morphine before being sent to hospital. When I saw him at 9 p.m. he was free from pain, his belly was soft, and there was little tenderness on pressure. His temperature and pulse were normal. I advised against operating, and did not see him for twelve hours. He then complained that the symptoms were returning, and he was operated on two hours later and a small perforation with comparatively little free fluid discovered. The temperature in the patients who were seen early, was normal, but in no case was the temperature elevated above 37.8° C. The pulse-rate varied from normal to 120. Absence of liver dulness was not noted, and this as a sign appears to be of little value, as an apparent absence of liver dulness is so often seen where there is only a distended colon. Distension was not marked, even in the late cases.

A diagnosis of perforated gastric or duodenal ulcer was made in seven cases. One was diagnosed as appendicitis, and the first incision was made over the appendix. The incision in the cases of recognized perforation was made a little to the right of the mid-line, and extended from the xiphi-sternum to the umbilicus. When the peritoneum was incised, sour-smelling but non-offensive gas escaped, and whilst here I might mention a point I read some time ago. Before cutting the peritoneum, the space above it is filled with saline solution, and if there is any free gas, no matter how little, it will be noticed to bubble through the saline fluid when the peritoneum is nicked. The escape of gas is a certain sign that a hollow viscus is ruptured, and the smell or its absence will suggest the organ affected. There was no free blood in the cavity in any of the cases, and it was surprising to note how little blood there was around the perforation. In seven of my cases the hole appeared as if it had been punched out, and the openings varied in size from one through which a pen-handle could be passed to one through which a knitting-needle would hardly go. The eighth had a large, ragged opening and hardly any induration. In the others an extensive area of induration surrounded the opening. The perforations were all on the anterior wall, and all within 1.75 cm. (1½ in.) of the pylorus, five on the stomach and three on the duodenal side. In the duodenal cases the escaped fluid was bile-stained and not nearly in the same quantity as in the stomach cases.

Pieces of brawn and tea leaves were recovered in the cavity of the patient who had eaten well, and there was a distinct smell of beer from the youngest patient's cavity. As soon as the perforation was found, it was closed with two or three through-and-through chromic gut sutures, and then the ulcer was inverted with Lembert suturing. In one case a purse-string suture was used, but in the others interrupted or continuous sutures were applied transversely to the canal. This appears to be the best way, as with it there is less likelihood of narrow-

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on July 12, 1917.

ing the lumen. The inverting sutures were applied just outside the inflammory area, so that they would hold, and, of course, went down to the submucous layer. The abdominal cavity was carefully mopped out in every case, the right kidney pouch being specially attended to, and swabs on holders were passed down into the pelvis in the cases in which perforation had occurred many hours before and where the fluid had gravitated to the pelvis. A drain tube was placed in the right kidney pouch in each case. In four a drain was placed near the perforation, and in two tubes were passed into the pelvis and brought out through large stab wounds above the pubis.

Very little drainage came from any of the tubes, except the one in the right kidney pouch, and in the last two cases I only used this tube. All drainage was dispensed with at the end of forty-eight hours.

Posterior gastro-jejunostomy was done in three patients. This appears to be quite unnecessary unless one is satisfied that more than one ulcer exists, and at the same time believes in the physiological theory as to the cure of ulcer. Moreover, it prolongs the operation, and there is no reason why it should not be done at a subsequent date, when the patient is in a better condition to stand it, and only then if stenosis has occurred. Excision of the ulcer was not practised, nor did it appear to be necessary in any of the cases.

As regards after-treatment, heroin was given when pain was complained of, a continuous saline rectal infusion by the drip method was used, and after twelve hours drinks of albumin water were allowed. Peptonized milk and broth and Benger's food followed, and at the end of a week the patients were on light diet. Six were up and about on the seventeenth day.

Vomiting occurred in only one patient after operation. One patient hiccoughed almost continuously for fifty hours, and nothing appeared to give him any relief. The pressure of the rubber tube on the diaphragm may have been the cause, for the hiccoughing ceased shortly after its removal.

The last case had a very bad complication. He was a thin, unhealthy subject, with little reparative power. A bad bronchitis developed after the anæsthetic, and some suppuration occurred along the wound. On the seventh day the silk-worm gut sutures cut right through and the catgut sutures gave way. The wound opened up widely, exposing an area of the left lobe of the liver as big almost as the palm of the hand, and a portion of the stomach with the ulcer area. I did in this case what I had not done previously, that is, when I had closed the perforation I covered it with a piece of omentum. The result was that on the eighth day the stomach and liver ascended and the omentum dragged the transverse colon into the wound. A few hours later a coil of small intestine was showing. He was again anæsthetized. The organs were separated from the abdominal wall and the wound closed with through-and-through silk-worm gut sutures. Suppuration was still occurring, and the wound healed poorly. This patient is going to have a hernia and much trouble with adhesions.

It will be noted that in all these cases there were straight out perforations on the anterior surface. I have seen a few cases that were diagnosed as leaking ulcers on the posterior wall. All these patients recovered without operation, so that there was no verification of the diagnosis.

The shortest time that elapsed from the time of perforation to operation was three hours, and the longest 38 hours.

All of my patients, with the exception of the last, now enjoy fair health, without any marked symptoms of indigestion.

The patient aged 49 years was operated on nearly four years ago. I thought at the time that the perforation might have been through a cancer, but I heard from him a fortnight ago, and he is quite well.

## Reports of Cases.

### A CASE OF SPLENECTOMY.[1]

By **Howard Bullock, M.B., Ch.M. (Syd.), F.R.C.S. (Eng.),**
*Honorary Assistant Surgeon, St. Vincent's Hospital, Sydney; Honorary Assistant Surgeon, Sydney Hospital; Senior Surgeon, Renwick Hospital for Infants, Sydney.*

I wish to bring under your notice this evening the case of a patient operated upon by me in May, 1917, for enlarged spleen.

The patient, T.C., 45 years of age, a spinster, a *virgo intacta*, has lived in the Bombala district all her life.

Her mother and father are both dead. The former died at 69 years of age, the latter at 70 years. There are eight other children, all healthy. There is no history of syphilis, malaria, *potus* or any family splenic trouble.

The patient was quite well till 15 months ago, when she was taken ill suddenly with stabbing pain under the left breast, suggestive of pleuritic trouble, and on account of which she was laid up for two weeks.

Shortly after convalescing varicose veins in the legs, which she had had for about seven years, started to swell and cause her pain. The left leg was worse. Suddenly a tumour appeared under the left costal margin, and with its appearance the pain in the legs vanished and the veins subsided.

Then her appetite became capricious, she had nausea after meals, and started to lose weight and colour.

When she came under my notice an obviously enlarged spleen could be felt extending well below the umbilicus. Dr. Walton Smith saw her, examined the blood, and diagnosed the condition as splenic anæmia, and advised operation.

Most authorities recommend Bevan's incision for splenectomy, which, as you know, is really a modified para-rectal incision on the left side, but I used a Sprengel's transverse incision, cutting the left rectus muscles completely across and portion of the right rectus muscle also, which gave remarkedly good access to the organ. Moschsowitz[1] writes in glowing terms of this incision for work on the gall-bladder and stomach, but I can find no instance of it having been used for a case of splenectomy.

From an anatomical point of view, the incision is thoroughly sound, as the muscle fibres and those of the aponeurosis run practically transversely in this region, and these need only be split to extend it. Thus it has everything in its favour to recommend its adoption for these cases and for upper abdominal work.

The operation went off very successfully, all adhesions and vessels being carefully tied *seriatim*.

[1] Read at a Meeting of the New South Wales Branch of the British Medical Association on July 13, 1917.

The convalescence was uneventful, the pulse did not go beyond 90, and the temperature for a few days rose to 37.2° C. (99.2° F.). The patient improved immensely from the first day; the appetite returned, the colour improved, and by the twenty-first day after the operation she had left the hospital.

She is now remarkably well, but I regret to say she was too nervous to present herself for inspection this evening.

Osler[2] reports six cases of splenectomy for splenic anæmia, with three deaths.

Giffin[3] reports 27 cases of splenectomy for the same trouble, with three operative deaths and eight in ten years.

W. J. Mayo[4] reports 58 cases, with five deaths—two from hæmorrhage and sepsis.

Johnston[5] reports 61 cases, with twelve deaths.

### References.


[1] Moschsowitz—*Annals of Surgery*, September, 1916.
[2] Osler's "Principles and Practice of Medicine," 1914.
[3] Giffin—*Annals of Surgery*, August, 1915.
[4] W. J. Mayo—*Annals of Surgery*, August, 1915.
[5] Johnston—*Annals of Surgery*, July, 1908.


## NOTES ON THE DIFFERENTIAL DIAGNOSIS AND PATHOLOGY.


By **P. E. Walton Smith, M.B. (Syd.), M.R.C.P. (Lond.),**
*Pathologist, No. 4 Australian General Hospital; Honorary Assistant Physician, Royal Prince Alfred Hospital, Sydney.*


Splenic anæmia, or Banti's disease, can be divided into three groups, according to the severity of the condition.

The first group comprises those cases where there is enlargement of the spleen without symptoms; the second, cases of splenomegaly with anæmia, and the third, cases with splenomegaly, anæmia, jaundice and ascites. By some authorities only those patients who come under the last heading are considered as suffering from Banti's disease.

Dr. Bullock's case belongs to the second group; the patient had splenomegaly with a mild degree of anæmia. A blood examination made on May 11, 1917, gave a red cell count of 4,000,000, a hæmoglobin value of 80%, a colour index of 1, a leucocyte count of 6,000, with 16.5% neutrophiles, no eosinophiles, 76% lymphocytes and 7.5% large mononuclears. The colour index was high, the general consensus of opinion is that in splenic anæmia there is a low colour index, although Emery gives it as 0.7 to 0.9. There is usually leucopenia, with relative lymphocytosis. This patient had a high percentage of lymphocytes, although the total number of leucocytes was not much below normal.

Six days after the operation the blood was again examined, and showed a marked alteration. There were now 5,000,000 erythrocytes, the hæmogloblin value was 100%, the colour index 1, leucocytes 11,400, with 49.5% neutrophiles, 1% eosinophiles, 45.5% lymphocytes and 4% large mononuclears. On June 1 the red cell count was still 5,000,000, the hæmoglobin value 100%, with a leucocyte count of 9,600, neutrophiles 50%, eosinophiles 1%, lymphocytes 45.8%, large mononuclears 3%, mast cells 0.2% (500 cells counted). The effect of splenectomy in a normal animal is to produce an anæmia which is at its greatest intensity three to six weeks after excision, after which the blood gradually returns to normal. A leucocytosis occurs within 24 hours. In splenic anæmia the blood begins to improve shortly after the removal of the spleen. McKendrick records a case in a girl, æt. 8 years, in whom there was a very rapid increase in the formation of red blood corpuscles within twenty-four hours of the operation.

The spleen weighed 1,600 grammes (3 lbs. 9 ozs.); the capsule was slightly thickened; there was no peri-splenitis and no evidence of infarction.

Sections of the spleen showed a generalized hyperplasia of the lymphoid tissue, so that the identity of the Malpighian bodies was lost. Stitt considers that this is the characteristic feature of the spleen in Banti's disease, and that those cases in which fibrosis is a marked feature (and which other writers consider the essential lesion, together with atrophy of the Malpighian bodies) are really syphilitic in origin. It may be that fibrosis developes in long standing cases. There was no fibrosis in the sections examined.

Cases of Hodgkin's disease, without apparent glandular involvement, may be mistaken for splenic anæmia, but in them the lesion in the spleen is totally different, as the Malpighian bodies become greatly enlarged and are seen by the naked eye as suet-like masses surrounded by pigment. It is possible in the light of recent bacteriological investigations that there is some direct bacteriological relationship between Hodgkin's disease, splenic anæmia and other forms of splenomegaly, from the fact that in two spleens removed from patients suffering with splenic anæmia, pure cultures of diphtheroid organism were obtained. These organisms appeared to be identical with the *bacillus hodgkini*. Primary splenomegaly of the Gaucher type is a distinct disease. It commences, as a rule, early in life, and may affect several members of the same family. The liver becomes enlarged without jaundice or ascites, and there is nothing characteristic in the blood. Microscopically the spleen is seen to owe its enlargement to the presence of numbers of large endothelial cells, with small nuclei and peculiar hyaline cytoplasm. By some authors the condition is, on this account, classed with the endotheliomata, but the disease has nothing in common with malignancy, as it runs a chronic course, and death is usually due to some intercurrent affection.

Splenic anæmia is the one disease associated with splenomegaly, in which splenectomy is most beneficial, and the difference it has made to Dr. Bullock's patient can best be appreciated by those who saw her at the time of operation and in her present condition of well-being.

## TRUSTEE COMPANIES.

Medical practitioners have the reputation of being bad business men, but even good business men recognize advantages of seeking the assistance of well-established trustee companies in the ordinary matters of personal or family finance, rather than that of an amateur trustee. There are two firms that invite the medical profession through our advertisements columns to make use of the facilities which they are able to offer, and the acknowledged reputation of both companies will guarantee the soundness of the propositions they place before our readers. These companies act as executors, trustees or attorneys under power for absentees. The experience they have gained is available for their clients in the selection of investments. Those who have entrusted the care of their affairs to these companies have learnt that economy is effected thereby, and that they have been saved a great deal of unnecessary worry and not a little unnecessary labour. It is claimed by trust companies that secrecy is better preserved by them than by individuals, and, inasmuch as the officers of these companies are under bond not to divulge the affairs of their clients, the claim is certainly well founded.

The Medical Journal of Australia.

SATURDAY, AUGUST 25, 1917.

## Repatriation.

A Bill was presented to the Senate of the Parliament of the Commonwealth on July 18, 1917, for the purpose of making provision for the repatriation of Australian soldiers. The Bill, as was pointed out by Senator Millen, the Vice-President of the Executive Council, is a machinery bill, but the machinery has been constructed on a definite policy. This policy was outlined in a most lucid and thorough manner by Senator Millen. It has now received the sanction of the Senate and the second reading was reached in the House of Representatives on August 8, 1917. Its aims and objects are admirable, and must meet with universal approval. The Commonwealth Government recognizes its duty to grant assistance and benefit to Australian soldiers upon their discharge from service and to the children of deceased or incapacitated soldiers. The Minister has decided to extend the scope of the measure to include the granting of benefits to the widows of deceased soldiers with children. Parliament agreed without discussion on the general principles underlying the measure, and accepted, after some discussion, the Government's proposals in regard to the methods of administration. With these general matters the medical profession has no special concern, beyond the concern which attaches to all citizens of the Commonwealth. There are, however, aspects of the question of repatriation which are to some extent medical, and it is therefore necessary for us to call attention to the proposals that deal with these chapters and to attempt to analyse the soundness of the principles on which the provisions are based. As this subject is a very large one, and many details will have to be discussed, only one question of fundamental importance will be raised at present, and the criticism of other matters will be deferred to some future occasion.

Senator Millen distinguished sharply between the means that would have to be taken to aid those who return from the front sound and well, and the means that would have to be taken to aid those whose efficiency is impaired as a result of wounds or illnesses. For the latter he suggested that elementary training in various vocations would be given in curative workshops attached to the hospitals. Advanced training would be available either at Government institutions which would have to be established for the purpose, or by arrangement with private employers. The Government would undertake to supplement the men's wage to bring it up to the recognized level for the particular occupation selected. Special homes or other provision would be required for the totally incapacitated. Senator Millen made a passing allusion to the possibility that a few men included in this class might be able to follow some occupation. In order to understand the situation it is necessary to realize that the Australian Soldiers' Repatriation Bill is a civil measure, and that the assistance it may afford will commence after the soldier has been discharged. Under ordinary circumstances a returned soldier is discharged six months after his arrival in Australia. The military authority loses all control over the man at the expiration of this term, and any treatment provided can only be offered, but not enforced. In Great Britain the incapacitated soldier is not discharged until it has been ascertained that medical science can do nothing more to improve his condition and to render him fitter for the struggle in civil life. The object of this is to keep the men under discipline as long as treatment is likely to have a beneficial effect, and to guarantee that the treatment shall be suitable and efficient. Both the medical officer and the patient are responsible to the authority, and in a well-organized service defects can be reduced to a minimum. Elementary and even advanced training can be provided during the period of treatment, and the workshops or training institutions can be administered by the military authority. Complaints have been made in the past that there is no semblance of discipline exercised over returned soldiers in some of the Australian military hospitals. If these complaints be well founded, the remedy is obvious.

In the men's interests, those responsible for the proper conduct of the military hospitals should see that they are humanely but strictly managed. By including in the civil repatriation scheme the partially incapacitated soldiers, whose wage-earning capacity may be improved by medical or surgical treatment, the chances of restoration of this power must be materially diminished. On the other hand, the Department of Defence could extend the period before discharge to conform with the British practice, and discharge could be effected when no further improvement could reasonably be expected. The best results would accrue under these conditions. There are two provisos: real military discipline must be established, and the patients must be prevented from taking French leave, often to return late at night under the influence of liquor; and the medical care must be in the hands of the most efficient physicians and surgeons, who would be directly responsible to the Department for the treatment, and must not be entrusted to men who owe their positions to private influence or personal favours. The soldiers who have fought and bled for the Empire should have the best, and only the best. We feel that under the practically voluntary provision of treatment and training included in the Government's repatriation scheme, permanent incapacity will become unduly prevalent.

## THE "MYSTERIOUS" DISEASE.

The New South Wales Branch of the British Medical Association has considered on two occasions recently a disease inappropriately called the "mysterious" disease. A little information is available concerning a series of cases of severe illness which has been observed at Broken Hill, Bourke and elsewhere, and it is around this sparce information that much speculation has accumulated. Drs. Cleland, Burton Bradley and E. Buckley have presented a survey of the facts and ideas concerning these cases. It may be of use if the position in regard to them were summarized concisely from three aspects.

In the first place, the question has to be asked: Is there but one disease from which the patients referred to have suffered? We are told that there were between March and June, 1917, no less than 57 cases of an illness characterized by certain symptoms. Drs. Cleland, Bradley and Buckley have indicated the course of the illness, and in the present issue we publish an article by Dr. Burnell, describing the cases observed by him at Broken Hill. A careful perusal of the reports reveals that neither the symptoms and course nor the pathological anatomical changes after death, as recorded, are sufficiently uniform or characteristic to justify a definite statement that this so-called "mysterious" disease is a single disease. At present the clinical descriptions do not help to determine this point. On the other hand, the pathological and especially the histological changes rather suggest that we are dealing with two or more affections. Dr. A. Breinl recorded in these pages on May 26, 1917, the interesting fact that in a case with similar clinical manifestations to those described in connexion with the cases at Bourke, there were well-marked cellular infiltration of the grey matter of the anterior horns of the spinal cord and other changes typical of acute anterior poliomyelitis. Dr. Cleland and his colleagues have also described similar changes in a case in New South Wales, and this finding has confirmation from Dr. A. W. Campbell. It must be accepted that these two cases were instances of acute anterior poliomyelitis, and the assumption that the causal organism in these cases was identical to that of the usual form of this disease, is permissible. Dr. Cleland has pointed out that cases of clinically recognizable infantile paralysis were not notified in the affected area during the period of the outbreak. It is inconceivable that 57 cases of acute disease should have occurred in a well-defined area without a large number of cases with paralysis persisting after recovery, were this disease uniformly anterior poliomyelitis.

In the second place, it may be asked: If the disease in the majority of the patients is not anterior poliomyelitis, is it a new, hitherto unknown, pathological condition, or is it a severe type of one or more recognized diseases? The only evidence in favour of the disease that affected some of the patients being a new condition is the finding by Drs. Cleland, Bradley and Buckley of an organism indistinguishable from a pasteurella. These observers admit that they do not place much significance on the finding of

this organism, and suggest that it may have been a secondary invader. Nevertheless, the organism was found in the blood stream and in the cerebro-spinal fluid of ten of the patients in Broken Hill. Otherwise, there is no evidence at all, either in the clinical manifestations or in the post-mortem changes that the condition is a new one.

The third aspect to which we would draw attention, is the possibility that some of the 57 patients may have been suffering from a septicæmia or toxæmia arising from some well-known infection. From the scant notes of the post-mortem examinations in a few cases, it is emphasized by Dr. Cleland that the characteristics of a septicæmia were wanting. Against this there was the opinion expressed by some of the speakers in the discussion on the case described by Dr. Litchfield that this case was possibly a meningococcal septicæmia. It is unnecessary at this stage to speculate as to the possible nature of the sources of a presumptive septicæmia or toxæmia, until it can be proven that we are dealing with an affection, or more than one affection, of this nature. For the present, it would be advisable to avoid the unsuitable designation "mysterious" disease, and we venture to suggest that until the uniformity of the pathological process in these cases can be demonstrated, the proposed term "cerebral fever" may also lead to confusion. Careful clinical observation, minute pathological examination and skilled laboratory investigation are needed to clear up the doubt surrounding these serious cases.

## DIPHTHERIA IMMUNITY.

The recognition of natural susceptibility and immunity to infective disease is of fundamental importance in the practical problem of dealing with epidemics. This knowledge has also a definite value in the handling of these diseases in individuals, families of individuals and small communities, such as schools. In the case of diphtheria, it has long been known that infants are rarely attacked, while children between the ages of two and five years are very prone to it. Recent researches have demonstrated that the immunity in infants and some children is due to a substance in the blood, or a quality of the blood, which leads to the neutralization of diphtheria toxin, when the blood and toxin are brought into contact. This substance or quality is usually known as "natural antitoxin." Various methods have been used for the purpose of measuring the antitoxic power of the blood. In 1913 B. Schick described a simple method of measuring the antitoxic power of the blood in the living subject. The intradermic injection of one-fiftieth of a minimal lethal dose of toxin is followed in a susceptible individual by a reaction, consisting in an area of redness and induration appearing in from 12 to 24 hours, and leaving a circumscribed area of brownish pigmentation which persists for from three to six weeks. When the blood contains natural or acquired antitoxin, no reaction follows. Dr. W. G. Savage[1] calls attention to the value of this test in the estimation of natural immunity. By its means the cause of the immunity to diphtheria in new-born infants can be explained. According to various observers the susceptibility to diphtheria is highest between the ages of from two to six years, and the test applied to a large number of children of these ages shows that over one-half of them have little or no antitoxin in their blood. He therefore assumed that, as the natural immunity disappears during the first few years of life, the loss of protection may be ascribed to the fact that the amount of antitoxin does not increase in infancy, and that the quantity sufficient to protect an infant, does not suffice for a child of two or three years. It appears that immunity or susceptibility, as measured by the Schick reaction, is frequently uniform in all members of one family. The test has been used as a guide in determining which children should be protected during an epidemic by a prophylactic injection of antitoxin. The duration of immunity conferred by a previous attack of diphtheria and by an injection of antitoxin, has been measured in this manner. Dr. Savage also calls attention to the value of the test in clearing up doubt in regard to the diagnosis in certain cases. Some interesting information concerning the degree of protection of bacillary carriers has been brought to light by this means.

## AS OTHERS SEE US.

The medical profession should be grateful to the members of the Eaglehawk Branch of the P.L.C. for having told us frankly what we are like. Human beings are notoriously bad judges of their own merits and demerits. We take comfort, however, in the reflection that we are to be suppressed—not eradicated.

According to a report in the daily press, the following resolution, referring to the position adopted by the Victorian Branch of the British Medical Association in connexion with the Friendly Society Lodges, has been passed by the members of the organization named above:—

> That this meeting expresses its disgust at the action of the medical unionists taking advantage of this time of national crisis to adopt the tactics of the extreme section of the I.W.W., by demanding higher wages, and threatening to strike if their demands are not conceded. That this meeting urges the National Government to take immediate action to suppress these enemies of society.

[1] *Public Health*, June, 1917.

# Abstracts from Current Medical Literature.

## OPHTHALMOLOGY.

### (58) The Argyll-Robertson Pupil.

John Dunn is not satisfied with the current theories explaining the pupillary reflexes or the Argyll-Robertson phenomenon, and finds many of them vague and inconclusive (*Archives Ophthal.*, May, 1917). He asks why should such elaborate mechanisms be accepted, when a much simpler and more direct pathway is available. This he is convinced is the ciliary ganglion. Light falls in the retina and stimulates the pigment layer, thence the ciliary region, and thence the sensory nerves to the ciliary ganglion, where efferent impulses are sent along its motor nerves to the *sphincter pupillae*. This he terms the primary light reflex. The path for the consensual light reflex is more complicated and twofold. Some fibres pass from the retina, nerve and tract to the geniculate and quadrigeminal regions, and thence to the third nerve nuclei; the other fibres pass from the retina to the optic nerves, chiasma, and tracts to the two sympathetic subthalamic ganglia. They stimulate the *dilator iridis*, through the sympathetic, and provide an equal sympathetic action on the dilator fibres of the two eyes. It is possible that the cones represent the terminal mechanism for the reception of impulses to the geniculate and quadrigeminal nuclei, and the rods represent those of the impulses to the subthalamic sympathetic ganglia. Lesion of the ciliary ganglion makes possible reflex iridoplegia, but it does not destroy the consensual reflex, nor, if confined to the sensory elements, leaving the motor element still connecting responsive to all impulses which reach it from the third nerve centres, does it prevent contraction of the iris in convergence and accommodation. The contraction of the pupils in tabes is due to the fact that the lesion in the ciliary ganglion attacks also the sensori-sympathetic connexions and thus inhibits the sympathetic *dilator iridis*. Later changes in tabes affect the motor portion of the ciliary ganglion, resulting in relative and sometimes unequal dilation of the two pupils and loss of all pupillary responses. The inequality of the pupils, the author considers, is a conclusive argument that the seat of the lesion in the Argyll-Robertson pupil is situated in the ciliary ganglia.

### (59) Glaucoma Simplex Without Perceptible Rise in Tension.

Harry S. Gradle describes the case of a single woman of 67 years, slightly myopic, whose sight had been failing for some years (*Archives Ophthal.*, March, 1917). The eyes appeared to be normal, the peripapillar retina was slightly hazy and fluffy, the edges of the disc were blurred in spots, and the disc was of a peculiar grey colour, with no cupping. The fields were normal. The tension in the right eye was slightly raised, but was easily reduced by massage. Repeated examination showed it to be not above the normal limits. In a second case, in a man 68 years old, shallow cupping of the discs ultimately developed. Treatment of this condition is of little avail. Mental and physical quiet are to be recommended, and weak miotics used. No operation should be performed. The author questions whether these cases should be regarded as a disease affecting the exchange of fluids, or is a disease of the optic nerve, or a combination of the two. He comes to the conclusions: (1) That the condition is a disease *sui generis*, neither a glaucoma nor a uveitis. (2) That it begins as a low-grade neuritis, leading to absorption of nerve fibres and formation of the caverns described by Schnabel. These changes lead at a later date to cupping. (3) That the intermittent rises in tension is due, not to interference with the outflow of fluid, but to oversecretion from absorption of toxins—a low grade of chronic uveitis. (4) That treatment has no influence on the disease. (5) That the cause is unknown—probably some systemic selective toxin for the optic nerve.

### (60) Dacryo-cysto-rhinostomy.

Alfred Wiener is an ardent advocate of the operation of dacryo-cysto-rhinostomy, and of the two routes prefers the external, which is simpler to perform and yields good results (*Archives Ophthal.*, May, 1917). In fact, he performs the Toti operation, with certain modifications. Local anæsthesia is preferred, and details are given for its accomplishment. The sac is exposed and separated from the lachrymal fossa. If found closely adherent, it is better to make an incision where the anterior wall is reflected on to the bone. The muco-periosteum is then removed from the lachrymal fossa, and with a 4 mm. trephine applied as low down in the lachrymal fossa as possible, a button of bone is removed. If possible, a flap (presumably of nasal mucous membrane) is made, and reflected back into the sac, or the mucous membrane is removed and the packing, with half strips of iodoform gauze saturated with vaseline, proceeded with. By passing a suture through the lower canaliculus (by means of an eyeletted probe) and attaching it to the upper end of the gauze strip, and having another suture attached to the lower end and coming out of the nostril, the gauze may be drawn up into the sac and later on changed. The anterior cut edge of the sac is now stitched with chromic acid gut to the adjacent periosteum, and the stem sutured. The author is satisfied with the results obtained in twelve cases.

### (61) Tarsectomy in Trachoma.

G. F. Suker gives useful indications for the operation of tarsectomy for trachoma. These may be summed up as intractability to treatment, damage to the cornea, and distortion of the tarsus (*Archives Ophthal.*, May, 1917). By using correct technique, bad results, such as entropion, lagophthalmos and ptosis, may be prevented. He emphasizes the following points: (1) Always have a 1 to 1.5 mm. strip of tarsus at the lid margin. (2) Begin the excision at the ciliary border. (3) Do not attempt to save the conjunctiva directly overlying the tarsus. (4) Do not excise more than just the tarsus. (5) Dissect the conjunctiva far enough to give a sufficiently large bare area without tension. (6) Do not excise the tarsus until the retrotarsal fold has been dissected. (7 and 8) Secure good approximation with a mattress suture. (9) Obliterate the "dead space" between lid and conjunctiva by two properly placed sutures, at the previous retrotarsal border of the tarsus. (10) Operate with a general anæsthetic. Tarsal clamps and two T-shaped forceps are employed. After the operation an ice-bag over the bandage lessens the reaction. After removal of the sutures astringents are advisable for the upper and lower conjunctiva.

### (62) Hypopituitarism Relieved by Glandular Treatment.

C. A. Elsberg and E. F. Krug consider that surgical treatment of hypopituitarism is unsatisfactory, and that hypodermic injections of glandular extracts should be tried first (*Archives Ophthal.*, March, 1917). Their patient, a woman of 23, ceased menstruating at 16, became drowsy and weak, suffered from thirst and polyuria, and partially lost her vision. Examination revealed optic pallor; bi-temporal hemianopsia; vision $^{20}/_{200}$ in each eye. A radiograph showed no change in the *sella turcica*. She was given pituitary gland tablets (0.18 gm.) three times a day for a month, but became worse. Right subtemporal decompression was then performed with temporary improvement, but two months later she was in a deplorable condition, weak and cyanosed. Treatment with pituitary extract was then begun, and one c.cm. of pituitrin injected hypodermically. Two days after the improvement was striking. In a week the patient was out of bed, and her general strength much improved. For the next two months she had a weekly injection, by which time the fields had enlarged and vision improved considerably. Two years later her vision was $^{12}/_{20}$. The optic discs were still pale.

## LARYNGOLOGY AND OTOLOGY.

### (63) Cystic Distension of Antrum Associated with Unilateral Blindness.

Pressure symptoms on the optic nerve, the result of simple distension of the nasal sinuses, apart from suppuration, are extremely rare. G. H. Pooley and G. Wilkinson have reported

the second case in their experience (*Journ. Laryng., Rhin., Otology*, March, 1917). The patient, a labourer aged 40, complained of failing sight of right eye, and examination, eleven weeks after onset, showed a white optic disc, somewhat swollen, with small vessels. Vision was less than $^6/_{60}$. The left disc was somewhat pale, vision with + 0.5 being $^6/_{12}$. There was no displacement of the right eye. A skiagram showed comparative opacity of the right antrum, and on puncture 10 c.cm. of straw-coloured fluid were obtained. On opening the antrum a thin-walled, collapsed cyst was found attached to the outer wall, close to the floor, the antral cavity smooth-walled and enlarged upward into the region of the anterior ethmoidal cells. There were no signs of bone absorption. Vision in the right eye remained unimproved, and a sudden unaccountable failure of vision in the left eye occurred, the vision a fortnight later being less than $^6/_{60}$; but it then began to improve. Seven weeks after the first operation the roof of the right antrum was removed by chisel and forceps, and the posterior ethmoidal cells opened up. No pus or fluid was found in them. The vision of the right eye did not profit, but that of the left eye rapidly improved, and three months later was practically normal. In the author's earlier case vision began to return in the affected eye within half an hour after the distended antrum was evacuated, then, however, the condition was detected before any optic neuritis had developed. The only other recorded cases of similar nature were published by Hajek, Benjamin, Hirsch (two cases), and Fullerton (three cases).

### (64) Stuttering.

A thorough physical examination for organic defects in the organs of speech and for nervous disorders, followed, if necessary, by treatment, should precede any corrective measures taken for the relief of stuttering. These latter measures, according to May K. Scripture and Eugene Jackson (*Laryngoscope*, February, 1917), include (1) proper posture, the whole body being held in a relaxed, comfortable and well-balanced position; (2) breathing exercises for increasing the lung capacity, as well as those to secure breath control in speaking. For the latter a deep, quick inhalation, without straining, with slightly opened mouth, and a slow exhalation with open mouth, are recommended. The next exercise is to utter, on the outgoing breath, firstly, vowels singly, then repeated, then the letters of the alphabet as far as possible, then sentences, gradually increasing in length until reinforcing breaths are required to complete them. The stutterer must be taught to breathe consciously when speaking until right breathing becomes a habit. (3) Phonation: proper inflection is of great help to the stutterer, and exaggerated inflection is advised at first. Singing scales, arpeggios and songs, also vowel drills, assist. (4) Articulation: the exact position of the speech organs for each vowel and consonant must be practised, by vowel drills; the vowels are lengthened and strengthened, first by themselves, then in words and sentences, the voiced and voiceless consonants learnt, final consonants enunciated, and all syllables given their full value. Reading and repeating poems, at first in company, then alone, give confidence; spontaneous work should be approached very gradually. Patients are advised to think always before they speak, and then enunciate slowly and calmly, being very careful of the first two words in each sentence, and being specially careful to lengthen the short vowels.

### (65) Atrophic Rhinitis.

Alterations in the nasal mucous membrane in atrophic rhinitis (ozæna) are not so profound as one would expect, says Wyatt Wingrave (*Proc. Roy. Soc. Med.*, Laryng. Sec., *Journ. Laryng., Rhin., Otology*, March, 1917). In the early stage, when the discharge is abundant, the mucosa merely shows a distinct increase in the lymphoid and vascular elements, though later, when atrophy and fœtor are pronounced, there is a shrinkage of the whole structure of the mucosa (including the nerves). The most striking feature is metaplasia of the surface epithelium—many layers of stratified squames, blocking the ducts, replace the columnar and ciliated cells, and this change may extend to the accessory sinuses and as far as the trachea, but never beyond the muco-cutaneous junction. There is never ulceration, necrosis nor bone involvement. Giant cell systems are never seen. The discharge is at first free, contains mucin, but never resembles pus. Later it becomes scanty, loses its mucin, and is adherent in crusts. Its dark colour may be due to anthracosis or chromogenic bacteria. Chemically the lipoids predominate, together with cell-globulin and keratin granules. Bacteria in great variety occur, chiefly saprophytes. Polymorphic acid and alcohol-fast bacilli are invariably present and closely resemble both types of tubercle bacilli. So far these bacilli have resisted orthodox methods of cultivation, but if a sterile emulsion, made from fœtid crusts, is mixed with Dorset egg medium, and "planted" with crusts, a thick felted growth slowly occurs. Five out of forced emulsion (five days), made from a typical case of ozæna, developed lesions indistinguishable from tubercle. The author concludes that certain organisms, morphologically and tinctorially resembling tubercle, are present in atrophic rhinitis, and these have not been found in any other nasal disorder.

### (66) Vincent's Disease of External Ear.

A girl, æt. 7, complained of pain in ear of three week's duration. The pinna was very tender. An inflamed area, covered by a thin, greyish, translucent membrane, with a definite irregular edge, extended over half the concha and into the meatus. A swabbing showed Vincent's spirilla and fusiform bacilli in abundance. The condition co-existed with a middle-ear suppuration of six month's duration. There was no pyrexia. Boracic fomentations dispersed the membrane in two days, and salicylic ointment completed the cure. Wingrave is quoted to the effect that the spirochaetes of Vincent's disease, when found in the nose, accessory sinuses, decayed teeth and middle ear, are almost invariably associated with pyogenic organisms. R. P. Mathers (*Journ. Laryng., Rhin. and Otology*, May, 1917).

### (67) Ætiology of Nose and Throat Conditions.

In a presidential address recently, Mark Hovell quotes Sir Richard Quain's statement that "most of the diseases of the throat have their origin below the midriff" (*Journ. of Laryng., Rhin., Otology*, January, 1917). The commonest cause of functional aphonia in women, at or past middle life, and in some cases in young women, is due, he states, to displacement or other affection of the uterus. Granular pharyngitis is not the result of improper voice production, but of derangement in either the gastro-intestinal or utero-vaginal tract, or in both, so that dyspepsia, constipation, diarrhœa, uterine displacement, and leucorrhœa all require serious treatment if the throat condition is to be cured. Nasal obstruction also, due merely to hypertrophy of soft tissue, and not relieved by cauterization, is in many instances the result of gastro-intestinal irritation overlooked or insufficiently treated.

### (68) Acidosis Following Nose and Ear Operations.

Varying degrees of acidosis follow anæsthesia in about 20% of cases in children, and in individuals predisposed by chronic infections, age, etc. Three degrees of severity are described by Ira Frank (*Annals Otol., Rhinol. and Laryngol.*, December, 1916). (1) Subacidosis: acetonuria and rapid pulse. (2) Acidosis: diacetic acid in urine, rapid pulse and respiration, and drowsiness. (3) Acid intoxication. A lessened carbohydrate diet, and shortening as far as possible the starvation period prior to operation, the use of nitrous oxide or short ether anæsthesia, and resumption of carbohydrate diet at earliest hour after operation, all tend to lessen the degree of, or to prevent, acidosis. Glucose, usually combined with sod. bicarbonate, *per os* or *rectum*, or in severe cases intravenously, speedily clears up the condition.

### (69) Epistaxis in Hæmophilia.

Hereditary defect in the platelets is held by G. R. Minot and R. I. Lee (*Arch. Intern. Med.*, October, 1916) to be the cause of the poor clotting-property of the blood in hæmophilia. The other elements are practically normal, as also is the number of platelets, thus differing from purpura, in which the number is decreased. Even if 75 times the normal number of such platelets be added to hæmophilic plasma, the coagulation time is not appreciably lessened.

# British Medical Association News.

## SCIENTIFIC.

A meeting of the New South Wales Branch was held at the B.M.A. Building, 30-34 Elizabeth Street, Sydney, on July 13, 1917, Dr. R. Gordon Craig, the President, in the chair.

Dr. Howard Bullock read the notes of a case of splenic anæmia, in which he had performed splenectomy, and exhibited the specimen removed. He also read a report by Dr. P. E. Walton Smith on the pathological findings. These two communications will be found on page 163 *et seq.*

Dr. J. Flynn congratulated Dr. Bullock on the success of his treatment. He assumed that Banti's syndrome was a primary splenic affection, as contrasted with those conditions in which the spleen was affected secondarily. Many authors had described the spleen as a ductless gland, but it was not a gland at all, since its structure did not contain epithelial cells, nor was there any evidence of secretion from it. Gray had published, many years before, a treatise on the structure and functions of the spleen, to which there was very little to be added. Seeing that it had few diseases of its own, almost all its ailments were of a secondary nature. As it enlarged in many toxic and infective conditions, it was probable that it was in some way associated with the protective mechanism of the organism. Thus it enlarged in disturbances of metabolism, *e.g.*, rickets, in infections, *e.g.*, malaria and syphilis, and in primary blood conditions, *e.g.*, leukæmia. But it was the merit of the Florentine pathologist, Banti, to pick up this particular group of enlargements, to segregate them from the groups already alluded to, and to show that there was a definite sequence of events in three phases, namely, first stage, simple enlargement; second stage, enlargement plus anæmia; third stage, enlargement plus anæmia plus cirrhosis of the liver. These stages had more than an academic interest. The removal of the spleen should be undertaken during the second stage, to save the liver. Dr. Flynn referred to the work of Paucot, of Lille, dealing with the effect of opotherapy in splenomegaly. Paucot claimed to have obtained a cure in almost every case by giving the pulp of fresh beef spleen.

Dr. W. F. Litchfield referred to a child who had suffered from Banti's disease for about ten years. He had seen the child a month before. There was not much anæmia. There was leucopenia and a relative increase in the number of lymphocytes. This was the first case he had seen in a child, although lymphatic leukæmia was common.

Dr. Archie Aspinall spoke of three post-mortem examinations he had carried out in 1907 in cases of splenic affections. In all of them there was marked cirrhosis of the liver. The blood count had revealed that there was no great alteration in the cellular elements. Some improvement had followed the use of X-rays. He also referred to a patient who had been subjected to operation for the removal of the spleen through a left rectus incision. The operation had been abandoned on account of very extensive adhesions. He thought that, had the method adopted by Dr. Bullock been applied in this case, the result might have been different.

Dr. J. C. Edwards discussed the evidence of the spleen being the seat of primary disturbance in Banti's disease. He pointed out that improvement was frequently maintained as long as X-rays were continued, but that all the patients eventually died within a year. It was necessary to use hard rays. In view of the results of radiological and other conservative treatment, he was inclined to advocate splenectomy.

Dr. R. Gordon Craig stated that he had not used a transverse incision alone in these cases. The incision he had employed had been along the border of the left rectus muscle, and to this he had added a transverse incision, thus making a triangular flap. More room could be obtained by dissecting the skin as far as the costal cartilages. In discussing the questions why the spleen was enlarged in Banti's disease, and how its removal gave rise to improvement, he referred to the theory that the spleen effected a breaking-down of used-up red cells. There appeared to be an over-destruction of red cells in Banti's disease, and removal apparently limited this destruction. He recognized that this explanation was probably too obvious to be true.

In his reply, Dr. Bullock informed Dr. Flynn that it was suggested, as far as his case was concerned, that cirrhosis of the liver had not had time to develope. He held the opinion that splenectomy was only of value when the spleen was the seat of the primary trouble. He agreed with Dr. Aspinall that dense adhesions constituted a great difficulty in the removal of the spleen. It might be impossible to remove the spleen on this account, and the surgeon had no alternative but to close the abdomen. In conclusion, he referred to the favourable reports published in America of splenectomy in the treatment of pernicious anæmia.

Dr. Archie Aspinall read some notes on a case of fractured skull. Dr. Aspinall's paper will be published in next week's issue.

Dr. J. Crawford Robertson stated that he had seen quite a number of cases of fractured skull in the Garrison Hospital. In dealing with the diagnosis, he advocated making a lumbar puncture in all doubtful cases. If the cerebro-spinal fluid were found to be blood-stained, or to be under increased pressure, the diagnosis of fracture was made, and a decompression operation undertaken. He was in favour of opening the *dura* in every case. He was inclined to the opinion that the convalescence was thereby shortened. It was not possible at present to determine whether these patients would develope epilepsy in after life. He had used urotropin to relieve the headache, with good results.

Dr. W. F. Litchfield suggested that decompression was usually carried out as a last resort. He referred to a case in which a small incision had been made into the dura and the cerebro-spinal fluid, under increased pressure, had escaped quite a foot into the air. If too much cerebro-spinal fluid were withdrawn, there was a danger that the medulla might be drawn downwards, and sudden death might occur. In one case he had seen extreme collapse in a boy, after the removal of cerebro-spinal fluid.

Dr. R. Gordon Craig called attention to the fact that Dr. Aspinall had not trephined, but had used the gap in the bone to insert an elevator to raise the depressed bone. Mayo had regarded it as inadvisable in all cases. He had taken advantage of the gap in several cases, and advocated this method whenever it was found practicable. In regard the question of opening the *dura*, he stated that no improvement usually followed in cases of fracture of the base of the skull, and in tumour cases in civil life, without an incision being made. On the other hand, when there was a lacerated, dirty wound, the *dura* should not be incised unless there was strong evidence of increased intracranial pressure. It was usually very difficult to re-unite the *dura mater* after incision. There was a grave danger of spreading sepsis. Jacksonian epilepsy developed as a result of the stimulation of the motor cortex. In some cases the least touch produced a fit. He had obtained excellent results from the interposition of a membrane of rubber tissue between the brain and the inner table of the skull. He cited cases in illustration of his contention that patients with septic infection were the most likely to develope epilepsy.

Dr. D. Kelly read a paper on perforated gastric and duodenal ulcers (see page 162).

Dr. Archie Aspinall asked Dr. Kelly whether his patients were able to lie down before the operation was performed. One of his own patients had been forced to sit up until the operation was performed. The perforation had taken place after the patient had drunk beer. The abdomen was quite rigid, the liver dulness was diminished, and there was crepitation in the lower portion of the chest. On opening the abdomen he had found the cavity full of the stomach contents. The site of perforation was not discovered. It was not on the anterior wall. The abdominal cavity was mopped out and drained. The patient did well for about a fortnight, when a sub-phrenic abscess developed. In another patient, who had been under the care of Dr. Binney, the rupture had taken place suddenly. The anæsthetic had to be given while the patient was sitting up.

Dr. Eric Jeffrey spoke of a case in which the onset was sudden. There was board-like rigidity of the abdomen. There was no diminution of the liver dulness. At the operation a tight contraction of the intestine, as was met with in enterospasm, was discovered. There was a hernia, but

it was not strangulated. and nothing else was found. He spoke of other cases without objective signs being discovered at the operation. In one case the temperature was rising, and at the laparotomy perforation of a duodenal ulcer was discovered. These cases had taught him that there was considerable difficulty in the diagnosis of the causes of acute abdominal conditions.

Dr. J. Crawford Robertson referred to a case of ruptured aortic aneurysm simulating ruptured gastric ulcer. The patient was an internee at the German camp.

Dr. R. Gordon Craig was surprised that Dr Kelly had found sub-phrenic abscess complicating perforated gastric ulcer rare. In his experience it was not at all uncommon. He held that the localization of sub-phrenic abscess was one of the most difficult problems in surgery. In regard to the situation of the ulcer, he recited the details of the case of a man who had been ill for some considerable time. There were signs of a gastric leak, with spontaneous recovery. The patient eventually submitted to operation. A huge ulcer, through which two fingers could be passed with ease, was discovered well away from the pylorus, close to the coronary artery. It was impossible to resect the ulcer, and therefore the defect was closed over with the anterior portion of the liver. The patient died of inanition and septic absorption. Dr. Craig emphasized the fact that perforation due to cancer of the stomach was very rare, while perforation due to cancer of the large bowel was common. In the differential diagnosis, it was wise to remember that basal pleuro-pneumonia was often indistinguishable from an acute abdominal condition. There was no unanimity of opinion in regard to the advisability of performing gastro-enterostomy for perforated ulcer. He had found the operation of value for gastric ulcer situated close to the pylorus.

Dr. Kelly stated that the majority of his patients had assumed a semi-recumbent position. Referring to the case in which Dr. Aspinall had been unable to find the ulcer, he maintained that if the abdomen were full of gastric contents, there must have been a perforation in the anterior wall of the stomach. When the stomach was squeezed, the escape of its contents could be seen. The perforation could also be detected by inflating the stomach. Replying to Dr. Craig, he stated that he had been expecting a sub-phrenic abscess to develope in his patients, and considered himself very lucky that he had only experienced it once. He thought that the majority of surgeons did no advocate gastro-jejunostomy.

Dr. J. Burton Cleland read abstracts from some notes on the "mysterious disease" which had occurred in New South Wales in 1917. The notes had been prepared by Dr. Cleland, Dr. Burton Bradley and Dr. Emma Buckley. He prefaced his remarks by referring to the incidence of cerebro-spinal fever and acute anterior poliomyelitis during the second half of 1915, and 1916. In March, 1917, cases of a severe illness had been reported from Narrabri, Walgett, Collarendabri, Moree, Bourke, Broken Hill and Goondiwindi. The cases were regarded as atypical instances of cerebro-spinal meningitis. In some cases it was probable that they were of this nature. Though the mortality was high, the cerebro-spinal fluid obtained by lumbar puncture was under only moderate pressure, and yielded no cellular deposit. A visit had been paid to Broken Hill to investigate the disease, but by the time one of the authors had arrived, the patients had either died or convalesced. A week later two further fatal cases occurred, but since that date there had been no fresh cases. As the diagnosis of cerebro-spinal meningitis did not appear to be wholly satisfactory, material was collected for examination at Narrabri, Moree, Warren and Mungindi. Material was also obtained after death in two fatal cases in Sydney.

The authors suggested the term "cerebral fever" for the condition, in view of the fact that the cerebral symptoms were more marked than could be accounted for by an ordinary general toxæmia. They were able to trace 57 cases.

The symptoms and course of the disease were given as follows: The illness was preceded by one or two days' malaise, sometimes accompanied by vomiting. The onset was rapid, and the patient became stuporous, semi-comatose or irritable. The temperature reached 39° to 39.5° C. There was rigidity of the limbs, slight or definite head retraction and stiffness. Kernig's sign was frequently present. Tetanic convulsions at times preceded death, which occurred within a week of the onset. The temperature was usually high towards the end, and occasionally reached 41.7° C. In non-fatal cases there was a gradual and irregular fall of temperature and a slow improvement. A mental change had been noted in several cases. In one or two cases there was paresis of the muscles of the extremities. The authors stated that lumbar puncture was performed in 35 cases. In seven cases there was a "dry tap." The fluid was said to be clear in the majority of cases. In two cases it was found to be turbid at the second puncture.

In regard to the mortality, they stated that ten out of eleven persons, who were presumably suffering from this disease, died at Narrabri, and that twelve out of fifteen died at Broken Hill. In the Moree district five out of ten died, while in the Mungindi district seven out of eighteen died. The mortality was therefore varied from about 37% to about 90%. The total mortality was 61%. The majority of the patients were children. The youngest was two months of age and the oldest patient was 61 years. The patients at Broken Hill were all males, but in the other districts the distribution between the two sexes was about equal. The authors recorded that Dr. Fitzpatrick, of Mungindi, had formed the opinion that lumbar puncture was of marked therapeutic value. In other districts lumbar puncture did not appear to exercise a beneficial effect. In a few cases post-mortem examinations were conducted under unfavourable conditions. Enlargement of the spleen and hæmorrhages of the abdominal and thoracic organs were not reported in any case. On the other hand, enlarged Peyer's patches and mesenteric glands were noted in a few cases. In one, the lungs were in a state of red hepatization. No obvious meningitis was detected. Some fine, punctate hæmorrhages were present in one brain. There was definite œdema in another and congestion in three brains.

The authors proceeded to analyse the distribution of cerebro-spinal meningitis, acute anterior poliomyelitis and the disease under consideration. Of 112 cases of cerebro-spinal meningitis notified in New South Wales between February 1 and May 31, 1917, 57 occurred in the area affected by the "mysterious disease." Of the 57, they considered that at least 29 were cases of this disease. They continued as follows:—

> This leaves a balance of 55 cases for the rest of the State, of which 26 occurred in the metropolitan district. If to the 29 cases that we recognize as being probably this disease, can be added the others which presumably existed, but about which we have no information, and if the total thus obtained were subtracted from the 57 cases notified as cerebro-spinal meningitis in the districts affected, it would perhaps be found that the incidence of ordinary typical cases of cerebro-spinal meningitis in the affected districts was, having in view the density of population, proportional to other country districts of the State. As the other country districts unaffected by the mysterious disease were, according to the notifications, affected by scattered cases of cerebro-spinal meningitis and their presumed attendant "carriers," it is hard to see why, if the mysterious disease were a form of cerebro-spinal meningitis, a similar incidence of unusual cases had not occurred amongst them.

There were ten cases of anterior poliomyelitis but none occurred in the districts mentioned above.

The authors recorded that a Gram-negative bacillus was obtained from the blood of four of the Broken Hill patients and also from the meninges. This bacillus resembled a pasteurella or an aberrant type of the colon bacillus. It produced no results when injected into guinea-pigs or a monkey. They gave reasons for believing that it had no ætiological significance, and was possibly a secondary invader. While they recognized that it was not improbable that aberrant instances of meningococcal fever had been included among the cases of the disease in question, they stated that no bacteriological evidence was available that the disease was meningococcal fever. They also considered the question whether the condition could be due to the action of a neurotoxin allied to that of tetanus. They failed to obtain any experimental evidence in support of this suggestion. They then considered that the disease might be

either the cephalic type of anterior poliomyelitis or some allied and hitherto undifferentiated disease with somewhat similar symptoms. A child at Narrabri died of a typical attack after about a week's illness. No meningitis was found post-mortem, and there was no evidence of septicæmia. Sections of the medulla and of the upper part of the spinal cord showed various areas of round-cell infiltration, with destruction of nerve cells and congestion. The condition was very suggestive of the lesions found in acute anterior poliomyelitis. Dr. A. W. Campbell had examined the sections and confirmed their opinion. On the other hand, no unusual changes were discovered in the brain or spinal cord of a man who had died in Sydney, and whose case during life was considered to be one of cerebro-spinal fever, with clear cerebro-spinal fluid. They emphasized the doubt as to the real diagnosis in this case. Negative results were obtained on injecting an emulsion of parts of the brain and spinal cord into the peritoneal cavity of a monkey. They pointed out that the cerebro-spinal fluid from other cases failed to produce any ill-effect on monkeys, guinea-pigs and rabbits.

In conclusion, they stated that there were good grounds for considering that the disease might be a disease allied to acute anterior poliomyelitis, hitherto unrecognized. In support of this suggestion, they pointed out that there was an apparent absence of pure spinal cases of anterior poliomyelitis in the districts affected during the epidemic.

Dr. Cleland gave a summary of the post-mortem findings in seven cases, and some of the clinical notes of six cases. We propose to publish the clinical notes in a future issue.

Dr. Wolfe S. Brown referred to a patient whom he had had under treatment at Liverpool three years before. The patient had been sent in with a provisional diagnosis of idiopathic tetanus. There was no wound. They had been on the look-out for cerebro-spinal meningitis, especially as there was some stiffness of the neck. At the lumbar puncture 60 c.cm. of clear serum had been withdrawn. No bacteria were seen in the fluid. There had not been any antiserum used. The patient was dead on the following morning. A culture from the right side of the heart, the spleen and the cerebro-spinal fluid remained negative. There was some congestion of the brain and spinal cord, but no typical lesion was discovered. Later on they had seen a number of cases similar to those described by Dr. Cleland. Dr. Wolfe Brown also referred to a case of atypical cerebro-spinal meningitis, in which typical poliomyelitic symptoms developed at a late stage. In another case, under the care of Dr. Gillies, the cerebro-spinal fluid had been slightly turbid and had contained cells. In this case there were paralytic symptoms, and the meningococcus had been detected. He had seen twelve cases of cerebral irritation associated with fever. The spinal fluid had given a Noguchi reaction for globulin. One patient died six hours after admission. All the others recovered. Lumbar puncture had been carried out in each case.

Dr. W. F. Litchfield considered that Drs. Cleland and Bradley had been wise in suspending judgement in regard to the nature of these cases. He referred to a case from Bourke, the history of which he had related to the Branch in April (see *The Medical Journal of Australia*, May 5, 1917, page 384). In this case a Gram-negative coccus had been grown from the swabbings of the pharynx. He was unable to say whether this coccus was a meningococcus or whether it was identical with Dr. Cleland's Gram-negative organism. He thought that further investigation was necessary before the disease could be regarded as infantile paralysis or an allied condition. He referred to a case with a sudden onset and severe cerebral symptoms. The cerebro-spinal fluid was clear. He had given anti-meningococcal serum in doses of 30 c.cm., as he regarded it as a case of cerebro-spinal meningitis. The patient had made a good recovery. He was now inclined to think that it was not a case of cerebro-spinal meningitis. He raised the question whether it was possible for the cerebro-spinal fluid to be free of cells in cerebro-spinal meningitis. He regretted that the post mortem examination had not been very complete in the cases spoken of. They were not like cases of ordinary infantile paralysis, either of the spinal or of the cerebral type. In conclusion, he referred to the case of a baby at the Royal Prince Alfred Hospital, under the care of Dr. Mills. The baby had been admitted with symptoms of gastro-enteritis. Post mortem the substance of the brain was found to be intensely congested, and there were minute hæmorrhages scattered under the arachnoid. The brain substance throughout was pink. He was of opinion that the cause of this illness was the virus of anterior poliomyelitis.

Dr. R Gordon Craig stated that he was unaware that elaborate tests on monkeys were carried out at Microbiological Laboratory of the Health Department. Referring to the question of urotropin in the treatment of cerebro-spinal meningitis, of which Dr. Cleland had spoken, Dr. Craig stated that it had been proved conclusively that this substance was not an antiseptic, and that its antiseptic qualities were only developed when formaldehyde was split off by the action of acid.

Dr. Burton Bradley, replying for the three authors, made some remarks in regard to the tests carried out at the Bureau. The work had been carried out under some difficulty, as it had to be done in addition to the ordinary routine work, and their staff was limited. Dr. Bradley referred to the experimental work carried out with the poliomyelitis virus. He believed that they had been the first in Australia to produce experimental anterior polimyelitis in a monkey. He pointed out that the object of their communication was to record what had been done, so that the same ground would not have to be covered in the future, should the disease break out again.

## Naval and Military.

The 331st list of casualties, issued on August 11, 1917, contains the name of 880 officers, nurses and men. Of these, 14 officers and 345 men are dead, 5 officers and 173 men are wounded, one officer and 13 men are injured, 10 men are missing, 153 are prisoners of war, and 16 officers, 2 nurses and 147 men are ill in hospital. It is with great regret that we learn from the list that Captain Ronald Lennox Henderson, who was previously reported to have been wounded for the second time, died of wounds on July 31, 1917.

The following announcements have appeared in the *Commonwealth of Australia Gazette*, No. 129, of August 16, 1917:—

Australian Army Medical Corps.

Major R. Fowler is granted the temporary rank of Lieutenant-Colonel whilst commanding 2nd Australian Light Horse Field Ambulance. Dated 22nd January, 1917.

Army Medical Corps.

To be Major (temporarily)—

Captain (temporary Major) G. A. C. Douglas, Australian Army Medical Corps. Dated 16th July, 1917.

To be Captains—

Major C. J. Weedon, Australian Army Medical Corps. Dated 18th August, 1917.

Captain (provisional) A. V. Benson, Australian Army Medical Corps. Dated 12th July, 1917.

Captain (provisional) W. R. C. Beeston, Australian Army Medical Corps. Dated 31st July, 1917.

Honorary Major E. P. McDonnell, Australian Army Medical Corps Reserve. Dated 12th July, 1917.

Honorary Major F. G. Griffiths, Australian Army Medical Corps Reserve. Dated 19th July, 1917.

Honorary Captain C. W. Parramore, Australian Army Medical Corps Reserve. Dated 17th July, 1917.

Honorary Captain E. L. D. Parry, Australian Army Medical Corps Reserve. Dated 23rd July, 1917.

Honorary Captain E. N. Scott, Australian Army Medical Corps Reserve. Dated 28th July, 1917.

Honorary Captain H. T. Marsh, Australian Army Medical Corps Reserve. Dated 24th July, 1917.

Honorary Captain G. A. Brookes, Australian Army Medical Corps Reserve. Dated 10th July, 1917.

Honorary Captains G. S. Landon, D. S. Henderson, A. E. Taylor and H. B. Gill, Australian Army Medical Corps Reserve. Dated 18th August, 1917.

Honorary Captain A. S. Roe, Australian Army Medical Corps Reserve. Dated 13th July, 1917.
Alfred Roy Hudson. Dated 3rd July, 1917.
Jonathan Murray Maclean. Dated 16th July, 1917.
Charles James Taylor. Dated 19th July, 1917.
John Richards Harris. Dated 6th August, 1917.
William George Herbert Tregear, and Valentine Macdonald. Dated 18th August, 1917.

Appointments Terminated.

The appointments of the undermentioned Officers are terminated from dates stated opposite names:—

Lieutenant-Colonel H. S. Stacey. 19th July, 1917.
Lieutenant-Colonel H. N. Butler, D.S.O. 20th July, 1917.
Captain F. M. House. 9th July, 1917.
Captain H. D. L. Murray. 16th July, 1917.
Captain G. M. Parker. 18th July, 1917.
Captain P. G. McReddie. 19th July, 1917.
Captain N. N. Davis. 20th July, 1917.
Captain E. L. Fitzgerald. 2nd August, 1917.
Captain E. M. Fisher. 2nd August, 1917.
Captain A. Alcorn. 3rd August, 1917.
Captain R. A. Haste. 8th August, 1917.
Captain A. C. Oliver. 8th August, 1917.
Captain W. H. Roberts. 9th August, 1917.

## AUSTRALIAN ARMY MEDICAL CORPS COMFORTS FUND.

When we acceded to the request of the Australian Army Medical Corps Comforts Depôt to appeal for financial assistance, more especially for the purpose of providing the members of the Corps with Christmas cheer, we pointed out that approximately the sum of £500 would be needed, and that there was considerable urgency, inasmuch as the time for posting the parcels was approaching rapidly. Many weeks have elapsed, and all that we have been able to hand to the Depôt has been £34 8s. The cause is so good a one that it is unnecessary to adduce arguments why it should be supported. We trust that every medical practitioner in the Commonwealth who reads these lines will forward a small contribution straight away.

| | £ | s. | d. |
|---|---|---|---|
| Amount previously acknowledged .. .. | 33 | 7 | 0 |
| Dr. W. M. Helsham (Richmond, N.S.W.).. | 1 | 1 | 0 |

# Proceedings of the Australasian Medical Boards.

## NEW SOUTH WALES.

(*Continued from page* 152).

The Medical Board of New South Wales enquired on April 3, 1917, into a charge of alleged infamous conduct in a professional respect on the part of Dr. William George McClure. The respondent did not appear when called. Mr. Bathgate, appearing for the Crown, informed the Board that he had been told by Mr. Levy that the latter was appearing for Dr. McClure. Mr. Levy had attended a few days before, and had asked that his case might stand back. A telephone message was sent that morning to let him know that the case would be heard shortly. Mr. Levy had said that he was not appearing, as the doctor had not come in to consult him. He therefore proposed to have the case heard *ex parte*.

Arthur Kench, an officer of the Board of Health, stated that he knew Dr. William George McClure, and that he had served a duplicate of the complaint and a notice after both had been countersigned by Dr. Abbott, as Secretary of the Medical Board, on Dr. McClure.

Witness stated that since November, 1916, he had been making enquiries into the medical business carried on under the name of W. G. McClure, at 251 Elizabeth Street, Sydney. On the front of the building appeared in large letters "Hall of Health," and across the face of the building, running the whole length, were the words "Dr. W. G. McClure, M.D., L.M., L.R.C.S., England." On the left-hand window were the words "Dr. W. G. McClure, Specialist." On the next window were the words "Hall of Health," and on the panel of the doorway "Consult Dr. W. G. McClure, late of Canada. Health Officer and Police Surgeon, New Zealand, foremost specialist, legally qualified medical specialist in diseases of men and women, blood poison tests. Cures effected by latest scientific method."

Similar announcements were placed on other parts of the building. Witness stated that the business was carried on by one Samuel Freshman, who was not a legally qualified medical practitioner. On January 24, 1917, in company with a detective, he called at the premises and asked to see Dr. McClure. He had a conversation with Dr. Phillip Heymann. After conversation with Freshman and Heymann, he took some pamphlets from the typists' room. On the one pamphlet appeared the words "Write to-day, it is dangerous to delay. Price 1s." On the inside cover were the words "Dr. McClure's Sanatorium is centrally situated in the heart of the city and faces that lovely resort known as Hyde Park." There was also a photograph on the inside cover with the words "Eden House, 251 Elizabeth Street, Sydney." On the first page was the following: "Important notice. As a personal consultation and examination is essential in all cases, intending patients must call at the consulting rooms. Letters of enquiry will not be answered until after the above examination has been made and we are satisfied the complaint can be cured by correspondence. Hall of Health, 251 Elizabeth Street, Sydney, March, 1916. Dr. William George McClure, M.D., L.M., M.R.C.S., England, Successor to Dr. Walsh." Witness also called attention to the preface, which contained the following sentences: "It may be that the general practitioner lacks the courage, and it requires some courage, to face the facts, or more likely lacks the skill, and it requires much to master the situation. He has not the time nor the inclination, or the ability, to cope with these protean disorders, which certainly need expert knowledge, the result of long study, observation and research." Witness visited Dr. McClure's private residence at Cowper Street, Randwick, on January 25, 1917, in company with a detective. He saw Dr. McClure, and informed him that he was making investigations into the medical business carried on at 251 Elizabeth Street. Dr. McClure admitted that the Dr. McClure referred to in the pamphlet was himself. He was paid £3 3s. a week by Samuel Freshman for the use of his name in connexion with this advertising. He had not seen or interviewed a patient at Elizabeth Street for over two years. His daughter called every Friday for the money. Samuel Freshman did the dispensing during the time when Dr. McClure attended at 251 Elizabeth Street. He stated that Heymann was a partner of Freshman. Dr. McClure's name was used at the establishment by means of a rubber stamp facsimile of his signature. Dr. McClure stated that they had no authority to use his signature. He was 91 years of age.

Witness had seen a number of advertisements or "dodgers" posted in different portions of the city, and had had several of these selected. The majority were found in urinals and water-closets. Witness had never seen Dr. McClure enter the premises in Elizabeth Street. The majority of the patients were young men and soldiers.

Evidence was given to prove that William George McClure was a registered medical practitioner, and that the name of Samuel Freshman did not appear on the New South Wales Register.

Robert Horne, an Inspector of the Board of Health, stated that he had visited the premises of 251 Elizabeth Street on the morning of January 10, 1917. He was admitted by a woman and shown into a room where two men were sitting. One was a soldier. After a time he was taken into the consulting room and saw Dr. Heymann. He said that he was feeling run down and washed out. He asked Dr. Heymann what the treatment would cost, and he said £10, of which £3 would have to be paid at once. On a subsequent visit he paid the money. On each occasion there were several men, including soldiers, in the waiting room. Heymann stated that he did not fix the charges himself, but that they were fixed for him. He suggested giving a hypodermic injection, but witness objected.

Ernest Arthur Mortimer Fairbairn, a police constable, stated that he visited under instruction 251 Elizabeth Street

In the evening of January 22, 1917. He was shown into the waiting room, and after a quarter of an hour to twenty minutes passed into the doctor's room. He told the doctor that he was suffering from pains in the back and shooting pains from the groins. He had placed a piece of blood-stained cotton wool on his penis. He was asked if he slept well at night and said "No." The doctor told him that he was suffering from severe nerve trouble, and that he could give himself something to cure. He told the doctor that he was not prepared to go under treatment at the time, but that he would call again after a couple of days. He called at the premises on January 24, 1917. He stated that he was just about the same. He was told to take off his clothes. The doctor examined his chest, back and groin. He asked witness to lie on the surgical chair, and then proceeded to pass an instrument. He gave him some medicine which witness did not take. He paid £1 1s. for it.

Arthur Kench, on being recalled, stated that Samuel Freshman was the financial proprietor of the business carried on at 251 Elizabeth Street. He did not know whether there were any others interested in it.

Two witnesses were called to prove that the advertisements put in as evidence were collected in various parts of the city, and that the sketch of the premises was a fair representation of them.

On April 24, 1917, the Acting President of the New South Wales Medical Board had considered the charge against William George McClure, and the evidence in the matter. It appeared to the satisfaction of the Board that William George McClure had been guilty of infamous conduct in a professional respect. The Board therefore removed the name of William George McClure from the Register of legally qualified medical practitioners.

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The Medical Board considered on April 30, 1917, a charge against Dr. Philipp Heymann. Mr. G. R. Raves intimated that Dr. Heymann was not well enough to be present. He admitted that his client was a legally qualified medical practitioner, that Dr. William G. McClure was a legally qualified medical practitioner, and that Samuel Freshman was not a legally qualified medical practitioner.

Arthur Kench, an officer of the Board of Health, stated that he had been making investigations into the trading concern carried on by Samuel Freshman and others under the title of Dr. W. G. McClure at 251 Elizabeth Street, Sydney. He gave evidence in regard to the advertisements on and in the premises similar to the evidence given in the case against Dr. McClure.

On January 24, 1917, in company with a detective and an inspector, he visited the premises. The door was opened by a man named Richmond. Witness asked to see Dr. McClure, and was told that Dr. McClure was engaged with a patient. He was asked to step into the waiting room. He saw Dr. Heymann, who stated that Dr. McClure was away, sick. He stated that his name was Philipp Heymann, and that he was a legally qualified medical practitioner. In reply to further questions, Heymann had informed him that he was not the owner of the business, but that Samuel Freshman was. He declined to disclose what salary he was paid, but stated that he was paid weekly. Witness asked whether Freshman was on the premises, and received an affirmative reply. Heymann went into the dispensing room and shortly after Freshman appeared at the door. As he entered the door, the respondent said to Samuel Freshman: "Say that you pay me." Samuel Freshman stated that he had an interest in the business concerned, and that he paid the rent of the premises. The lease was in the name of Howard Freeman. Freshman declined to state the names of the partners in the concern. He admitted that he paid Dr. Heymann a salary, but declined to say what he was paying Dr. McClure. In reply to the question, "Why do you pass off as Dr. McClure?" Heymann stated that he did not. He admitted, however, that he did not inform the patients that he was not Dr. McClure, unless they asked him, and that some patients remained under the impression that they had been treated by Dr. McClure. Dr. Heymann stated that he had been working at the establishment for two years or a little longer, while Freshman had been Manager for seven or eight years. Freshman admitted that letters were signed "William G. McClure" by means of a stamp, and stated that Dr. McClure's permission was contained in the agreement. Witness asked Freshman who Dr. Walsh was, and pointed out that his name was on the lamp outside. Freshman said that it was not there, as Dr. Walsh had been dead for two years or more. Witness took him out to show the name on the lamp.

Witness also interviewed a man named Richmond, who had opened the door. He had stated that he was employed and paid by Dr. McClure, and that Dr. McClure treated patients. Witness also gave evidence in regard to patients who had been treated by Dr. Heymann, and in regard to cards referring to patients. He also repeated some of the evidence given in the McClure case.

Mr. Raves cross-examined Arthur Kench. Witness admitted that some of the signs had been removed from the premises recently. He had been instructed by the proper authorities to investigate the case. On being questioned whether he had any authority under the *Medical Practitioners Act*, or under any other Act, witness stated that he had acted under two or three different Acts, and that he held authority under several Acts. He had no special authority under the *Medical Practitioners Act*. Questioned as to whether he knew any doctors who had previously acted as *locum tenentes* for Dr. McClure, witness stated that he had never seen them, but that according to report there had been several doctors connected with the business. He understood that some years ago Dr. Rodgers, a member of the British Medical Association, had been at the establishment for a short time. Mr. Raves asked whether he knew that Dr. Rodgers, or his wife, had consulted the Secretary of the New South Wales Branch at the British Medical Association, whether there was any objection to his acting in this way. Witness replied that he did not know what Mrs. Rodgers had done three or four years before. Witness was also questioned in regard to Dr. Walsh.

A number of questions were put to witness in regard to the signature on the prescriptions and receipts given to patients. There was a receipt for 19s, given to a youth named Plummer. Witness stated that Plummer "was agitated that he was not going to get his money." He advised him to get a receipt for the money paid. Witness denied that this youth was frightened at the inspectors, detectives and others, in the sense that he replied "Yes" to every question. He was anxious whether he was being properly treated, and whether he was going to get his 19s. back. Witness had not seen a letter from Heymann to Plummer. Counsel attempted to make witness admit that Heymann had a strong German or Polish appearance and a strong foreign accent, and that it was unlikely that he would be mistaken for a man with a Scotch-sounding name. Witness stated that he spoke English very well, although he stuttered.

Witness admitted that he had not given Dr. Heymann any warning that objection was being taken to his conduct. Witness was present when Dr. Heymann called for his instruments and other things. Mr. Raves asked witness whether he remembered that Dr. Heymann said that he would have nothing more to do with it when he discovered what class of business it was, and whether Dr. Heymann was not surprised in the consulting room with the development of matters. Witness stated that Dr. Heymann was apparently excited that he had been discovered there. On being pressed whether the excitement was due to his being discovered there, or to his having discovered the nature of the business, Mr. Kench replied that he could not understand how Dr. Heymann could have been there for two years without having known the nature of the business. Counsel questioned whether Dr. Heymann had stated that he had been there two years, but witness said that he had taken a note to the effect that Dr. Heymann had told him, "I have been practising here for two years; that is the period that Dr. McClure has been away." He added that Dr. Heymann did not tell him that he had been deceived by Freshman. Mr. Raves asked a number of questions concerning the evidence witness had given about Dr. Heymann allowing people to believe that he was Dr. McClure. He admitted that Dr. Heymann's name was not

shown on the premises, and that he had not seen any advertisement containing Heymann's name.

Robert Horne, an Inspector attached to the Board of Health, gave an account of a visit paid by him to 251 Elizabeth Street, Sydney. His evidence was similar to that given in the McClure case, but contained more details. The witness stated most positively that Dr. Heymann did not give him a written receipt, nor a written prescription. In reply to Mr. Raves, witness denied that Dr. Heymann had told him about some remarkable cases he had dealt with by intravenous injections, and that he had said anything about having read a paper before the Australasian Medical Congress in Sydney in 1911. He did not tell him anything about the results obtained by injections of increasing doses of cacodylate of soda. In regard to the fee, witness stated that when he was discussing his inability to pay £10 Dr. Heymann had told him that the fees were not fixed by him; they were fixed by Dr. McClure. Mr. Raves put it to the witness that Dr. Heymann did not pretend to be Dr. McClure. Witness replied that that was the first intimation he had that he was not Dr. McClure. During the course of his attendance at the premises he had discovered that Dr. Heymann was not Dr. McClure.

Mr. Raves asked:—

> What I would like to know is this. As you admitted you were not suffering from any illness, and there was no question of cure, why, if you did not intend to undergo the suggested treatment, you called a second time and paid the £3? I mean, what did you tell the doctor? What was your explanation?

Witness replied:—

> I did not make any explanation. He took the money without asking. On the first visit he said he did not think there was much wrong with me, but he wanted £10 to fix me up.

Ernest Arthur Mortimer Fairbairn, a police constable, also gave evidence similar to that given in the McClure case.

Mr. Raves cross-examined witness. After asking some questions in regard to the fees, he asked whether he remembered anything about some cotton-wool which Dr. Heymann had removed from his penis. Dr. Heymann did not tell him that he was suffering from a urethral discharge. He did not decline to be treated for what the doctor told him he had discovered. Dr. Heymann did not give him a receipt or a prescription. Dr. Heymann did not tell him that he was Dr. McClure, but he had asked if Dr. McClure was in. He was there to be treated by Dr. McClure. Witness stated that he could not tell Mr. Raves whether he was a Scotchman or not; he was not much of a judge of nationalities. He did not appear to him to be like a foreigner. He had a terrible impediment in his speech. It was more than he could say whether he thought that he was an Englishman, a Scotchman or an Irishman. He did not know at the time that he was a German.

Arthur Charles Patton, an Inspector of the Board of Health, gave evidence of having removed two advertisements from a tin in the internal wall of the water-closet at West's Hotel in George Street. He also gave evidence as to a series of other advertisements fixed up in similar places.

Mr. Raves and Mr. Bathgate both addressed the Board.

The President read the findings of the Board to the following effect:—

> The New South Wales Medical Board has considered the charge against Philipp Heymann and the evidence in the matter, and it appears to the satisfaction of the Board that Philipp Heymann has been guilty of infamous conduct in a professional respect. The Board therefore now removes the name of the said Philipp Heymann from the Register of legally qualified medical practitioners.

Mr. Raves gave formal notice of appeal.

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On May 26, 1917, Mr. Justice Pring, Mr. Justice Gordon and Mr. Justice Ferguson heard the appeal of Dr. Thomas Benedict Clune in Banco. The appellant claimed that there was no evidence that he had been guilty of infamous conduct in a professional respect. After considering the arguments of both sides, the Court dismissed the appeal, with costs. In his judgement, Mr. Justice Pring expressed the opinion that, had the Medical Board come to any other conclusion than it did on the facts, it would have very seriously neglected its duty.

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## VENEREAL DISEASES.

The regulations under *The Public Health Act*, 1903 (Tasmania), and its amendments, dealing with venereal diseases have been issued by the Chief Secretary of Tasmania on August 14, 1917, and will come into operation on September 1, 1917. In view of the fact that the majority of these regulations are identical with the regulations issued in accordance with the provisions of *The Venereal Diseases Act*, 1916, of Victoria, which were published *in extenso* in *The Medical Journal of Australia* of May 26, 1917 (pp. 453-454), only a summary will be given in this place.

Clauses 1 and 2 are headed "Short Title" and "Definitions" respectively. Clause 3 is headed "Period During Which Medical Attention Necessary," and is identical with Clause 3 of the Victorian regulations. Clause 4, "Notices, Directions and Information to be given to Persons Suffering from Venereal Disease" is identical with Clause 4 (i) of the Victorian regulations. Clause 5, "Certificate of Cure of Venereal Disease or of Having Ceased to be Liable to Convey Infection Thereof," is identical with Clause 5 of the Victorian regulations save that the form referred to in sub-clause (8) is called No. 4 instead of D. Clause 6, "Certificate of Freedom from Venereal Disease," Clause 7, "Persons to Apprehend," and Clause 8, "Periods of Detention for Examination," are identical with the corresponding clauses of the Victorian regulations, save in regard to the numbering of forms and the reference to sections of the Act. There are no clauses corresponding to Clauses 9 and 10 of the Victorian regulations dealing with fees. Clause 9, "Return of Cases of Venereal Disease," and Clause 10, "Prevention of Infection," as well as Clause 13, "Penalties," are identical with Clauses 11, 12 and 13 of the Victorian regulations, save that the Chief Health Officer is named in the place of the Medical Inspector. Clauses 11 and 12 are as follows:—

*Hospitals Receiving State Aid to Treat Persons Suffering from Venereal Disease.*

11. The managers of all hospitals receiving aid from the State shall make such effective provision for the reception, accommodation, examination and treatment, free of charge, of persons suffering from venereal disease as the Minister, on recommendation of the Chief Health Officer, shall direct.

*Hobart Gaol to be a Place Where Persons Suffering from Venereal Disease may be Detained for Treatment.*

12. Such portion of the Hobart Gaol as the Minister, on the recommendation of the Chief Health Officer, shall, by notice in writing to the Superintendent thereof, direct shall be, and is hereby prescribed, as a place where persons suffering from any venereal disease may be detained for treatment under the provisions of the Act.

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# Correspondence.

## MILK AND MILD FOODS.

Sir,—Halliburton says "After all, cows' milk is but a poor substitute for human milk," so that we have to make the best of a bad job when attempting to hand feed an infant. In using cows' milk for this purpose the personal equation is everything. It includes the personal equation of the cow, the individual who draws the milk from the cow, the individual who sells the milk, the parent who looks after the feeding bottles and teats, and lastly, but not least, the baby.

Given a good cow's milk and cleanliness right though, it avails nothing if the baby cannot take the milk, and these cases do occur, in spite of all the dodges to alter the milk by dilution, lime water, barley water, etc.

As a general rule, however, a healthy baby can take cows' milk and do well upon it, provided the milk is always

fresh and cleanliness observed by all concerned. Until Lady Talbot started the fresh milk and small ice-chests for the babies of Melbourne, it was only the favoured few who could procure a reliable raw milk. If fresh cows' milk is not obtainable (I don't consider the daily suburban delivery fresh), then, in my humble opinion, there is no alternative other than to resort to the best of the artificial foods. Give them their due, they are a God-send in tropical climates. With the aid of fruit juice, raw beef juice, raw diluted egg albumin, etc., they can tide the child over the period until it can take older diet.

I think that the reason that there is such confusion of opinion amongst the mothers concerning the relative values of milk and prepared artificial foods is because in many of the cases it has been the presence of some gastric or bowel disturbance that has raised the question of an artificial food. The presence of disease affects the child's response to the ingestion of the artificial food concerned, whether it be a modified milk or a prepared food, and makes this food compare unfavourably with the food of another baby who is in good health.

Yours, etc.,

"SUBURBAN."

Melbourne, August 14, 1917.

## Medical Appointments.

In pursuance of the provisions of *The Insanity Act of* 1884, Dr. Francis Henry Vivian Voss has been appointed Medical Officer to the Rockhampton Reception House, South Australia.

Dr. John Joseph Luddy has been appointed a Medical Referee under *The Workers' Compensation Act Amendment Act of* 1916, for the whole of the State of Queensland.

The appointment of Dr. J. McDonald Allan as Medical Officer of Health, Moora, Western Australia, during the absence of Dr. W. S. Myles, has been approved.

Dr. Francis Wilfred Harlin has been appointed a Medical Referee under "The Workers' Compensation Act Amendment Act of 1916" for the whole of the State of Queensland.

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xvii.

Royal Hospital for Women, Paddington, Junior Resident Medical Officers.

## Medical Appointments.

IMPORTANT NOTICE.

**Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429 Strand, London, W.C.**

| Branch. | APPOINTMENTS. |
|---|---|
| TASMANIA.<br>(Hon. Sec., Bellerive, Tasmania.) | Medical Officers in all State-aided Hospitals in Tasmania. |
| VICTORIA.<br>(Hon. Sec., Medical Society Hall, East Melbourne.) | Brunswick Medical Institute.<br>Bendigo Medical Institute.<br>Prahran United F.S. Dispensary.<br>Australian Prudential Association Proprietary, Limited.<br>National Provident Association.<br>Life Insurance Company of Australia, Limited.<br>Mutual National Provident Club. |
| QUEENSLAND.<br>(Hon. Sec., B.M.A. Building, Adelaide Street, Brisbane.) | Medical Officers to the Selwyn Hospital, North Queensland.<br>Brisbane United Friendly Society Institute.<br>Warwick Hospital. |
| SOUTH AUSTRALIA.<br>(Hon. Sec., 3 North Terrace, Adelaide.) | The F.S. Medical Assoc., Incorp., Adelaide. |
| WESTERN AUSTRALIA.<br>(Hon. Sec., Health Department, Perth.) | All Contract Practice Appointments in Western Australia. |
| NEW SOUTH WALES.<br>(Hon. Sec., 30-34 Elizabeth Street, Sydney.) | Australian Natives' Association.<br>Balmain United F.S. Dispensary.<br>Canterbury United F.S. Dispensary.<br>Leichhardt and Petersham Dispensary.<br>M.U. Oddfellows' Med. Inst., Elizabeth Street, Sydney.<br>Marrickville United F.S. Dispensary.<br>N.S.W. Ambulance Association and Transport Brigade.<br>North Sydney United F.S.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society.<br>F.S. Lodges at Casino.<br>F.S. Lodges at Lithgow.<br>F.S. Lodges at Parramatta, Penrith, Auburn and Lidcombe.<br>Newcastle Collieries — Killingworth, Seaham Nos. 1 and 2, West Wallsend. |
| NEW ZEALAND: WELLINGTON DIVISION.<br>(Hon. Sec., Wellington.) | Friendly Society Lodges, Wellington, N.Z. |

## Diary for the Month.

Aug. 28.—N.S.W. Branch, B.M.A., Medical Politics Committee; Organization and Science Committee.
Aug. 29.—Vic. Branch, B.M.A., Council.
Aug. 30.—S. Aust. Branch, B.M.A., Branch.
Aug. 31.—N.S.W. Branch, B.M.A., Branch (Ordinary).
Sept. 5.—Vic. Branch, B.M.A., Branch.
Sept. 5.—Central Southern Med. Assoc. (N.S.W.).
Sept. 7.—Q. Branch, B.M.A., Branch.
Sept. 11.—Tas. Branch, B.M.A., Council and Branch.
Sept. 11.—N.S.W. Branch, B.M.A., Ethics Committee.
Sept. 13.—Vic Branch, B.M.A., Council.
Sept. 13.—N.S.W. Branch, B.M.A., last day for nomination of two Candidates for election of Federal Committee.
Sept. 14.—N.S.W. Branch, B.M.A., Clinical.
Sept. 14.—S. Aust. Branch, B.M.A., Council.
Sept. 18.—N.S.W. Branch, B.M.A., Executive and Finance Committee.
Sept. 19.—W. Aust. Branch, B.M.A., Branch.
Sept. 19.—South Sydney Med. Assoc. (N.S.W.).
Sept. 21.—Q. Branch, B.M.A., Council.

EDITORIAL NOTICES.

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